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SYMPOSIUM ON THE MECHANISM AND MANAGEMENT OF MENTAL DEFICIENCY IN INFANTS AND CHILDREN

PART II

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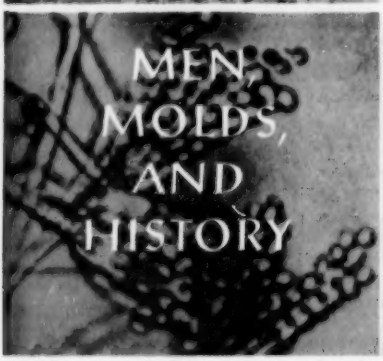

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Symposium on the Mechanism and Management of Mental Deficiency in Infants and Children

Part II

GUEST EDITOR—I. NEWTON KUGELMASS, M.D., Ph.D., Sc.D.

Chemical Therapy of Mentally Retarded Children

I. Newton Kugelmass, M.D., Ph.D., Sc.D.

CONSULTANT, DEPARTMENT OF HEALTH AND HOSPITALS, NEW YORK CITY

NEW YORK, N. Y.

CLINICAL TYPES OF MENTAL DEFICIENCY

Clinical Classification. Mental retardation is a symptom of an underlying systemic disorder; hence the diagnosis of amentia depends not only on the demonstration of intellectual deficiency but on the determination of the nature and degree of the defect responsible for the social inadequacy in the ament's environment. The mental defect may be endogenous, determined genetically, or exogenous, acquired environmentally. But the line of demarcation is not clear-cut, for congenital amentia may be due to environmental factors in utero while acquired amentia, manifest in early childhood, may be developmental in origin. Then too, the classification is further complicated by the degree of amentia, unrelated to the type of defect or the time of origin.

Psychometric tests reveal the child's functional performance at a particular time in comparison with that of his normal peers. They are orientation indicators of a child's general intelligence, giving a partial picture of his total behavior. A single test is of little moment unless duplicated under standard conditions. The tests should probe the mechanism of intellectual functioning, the nature of latent and manifest abilities, the pattern of the child's personality structure, the stability of his emotional reactions, the basis of individual motivation, the underlying attitudes toward himself, peers, family, strangers, and institutions, the *modus operandi* of achieving satisfaction, the modes of reaction to stress, and pertinent aspects of the psychodynamics of his behavior. Psychometric tests delineate the degree of mental deficiency if evaluated in terms of total behavior and social adjustment in the child's

Part I of this Symposium appeared in the February issue; part III will appear in the April issue.

TABLE I

Mechanism of Amentia (Jervis)

Endogenous amentia	Exogenous amentia
Single dominant gene	Infection
Dysostosis	Prenatal
Neurofibromatosis	Postnatal
Tuberculosis	
Single recessive gene	Chemical
Amaurosis	Poisoning
Cerebellar atrophy	Endocrinopathy
Diffuse sclerosis	Thyroid
Gargoylism	Pituitary
Hepatolenticular degeneration	Sex
Microcephaly	
Phenylpyruvic oligophrenia	Blood group
	Incompatibility
Multiple genes	Physical
	Irradiation
	Trauma
	Prenatal
	Paranatal
	Postnatal

natural environment. Penrose[53] demonstrated a rise in the incidence of amentia from 0.1 per cent in children less than 4 years old to about 2.5 per cent in the pubertal period, gradually diminishing to 0.5 per cent with age. The highest incidence during the school year merely reflects negative achievement in a competitive environment since school success depends on abstract thinking and adequate verbalization without the compensatory performance in other skills possible in later years.

The diagnostic appraisal requires comprehensive evaluation of the assets and liabilities revealed by capacities and limitations as a basis for effective treatment planning. The earlier the recognition of the underlying basis of the defect, the more effective the prevention of potential difficulties in the child's relationship to his environment. Many local and systemic disorders that decrease the rate of the child's development may simulate mental retardation. Normal intelligence does not preclude developmental lags from impaired vision and hearing, defective physical and motor function, disturbed environmental conditions, emotional tensions, specific deprivations, organic brain damage, infantile autism, or childhood schizophrenia.

Trainable, Educable Aments (Intelligence Quotient Greater Than 50). The infant moron develops at a relatively normal rate. His retardation may be so mild as to escape attention but his capacity is limited and his behavior disturbing. He is capable of adapting eye movements, staring with apparent attention, smiling on appropriate occasions, and responding to some stimulation. But he is restless and irritable, prone to digestive and nutritional disturbances. As a toddler he engages in senseless activity, rushing about the room, attracted by anything that catches his eye. Whenever he picks up something, he throws it down and darts off aimlessly on a rampage

of destruction. His attention cannot be held for a moment, and he is an impulsive creature devoid of natural controls over primitive behavior. The young ament plays in isolation, content with a few toys. If subdued, he may be accepted by his peers as long as he follows their dictates. It will take many years before he learns to play with others cooperatively or constructively. At the age of 5 he is about ready for play in the nursery school with younger children while his contemporaries are entering kindergarten. The mother senses developmental difficulty but shares the family optimism about his outgrowing it. Such opinions are unwarranted for there is no trend toward spontaneous improvement.

The childhood moron is educable in a special school for individual instruction and practical training. He can learn to read and write by the eighth year, do simple arithmetic problems, become familiar with elementary geography, history, civics, and other school subjects. But his maximum scholastic achievements rarely exceed the level of the sixth grade. Although educationally defective, he may have enough common sense for social adaptiveness. He can take care of himself physically, adapt himself to his surroundings, participate in games and sports, and can get along with his peers. If socially defective, he lacks common sense even though he may exhibit special ability in certain subjects or excel in certain activities. He cannot take care of himself, cannot adapt to his environment, cannot get along with playmates, cannot pass any practical performance tests. The moron mind presents a system of components whose permutations and combinations produce specific patterns. They determine the functional effectiveness of the being in every endeavor.

The adult moron has a social capacity ranging from the 7- to the 11-year level. He is unable to meet the ordinary demands of school and family life, and he requires supervision for his own protection and for the protection of others. He is capable of earning a living under favorable conditions but incapable of competing on equal terms or managing his own affairs with prudence. He shows obvious manifestations of biosocial immaturity. He may be trained for some simple task to support himself under sympathetic supervision, but in planning for his future, making recreational choices, engaging in personal relationships, and handling financial matters, he remains quite childish. He may become self-supporting, marry, and rear a family. But he will be subjected to repeated failures and frustrations in the significant areas of life's adjustments.

Trainable, Noneducable Aments (Intelligence Quotient 25 to 50). The infant ament of this group appears abnormal at birth. He is apathetic and listless and rarely moves unless disturbed. He shows no reaction to the mother's voice, to bright lights, or to colorful objects. When taken into his mother's arms, he does not respond with vigorous movements but remains limp and relaxed or rigid and fretful, his mouth wide open drooling saliva. With the best of care he achieves the first milestones of development, i.e., sitting, standing, walking, and talking, three years behind the normal. He rarely develops complete coordination but remains awkward and clumsy in his movements. Such motor infantilism exists quite apart from any neuromotor defect. Nervous instability is manifested by facial grimaces and involuntary movements, accentuated by seeing strangers and unaccustomed sights. His speech is delayed and defective. He cannot master the accepted techniques of interpersonal

relationships, but with personal supervision he may complete simple tasks, pursue ordinary recreation, take care of his daily routine, and even help about the house.

The imbecile child is trainable but not educable. Individualized instruction will take three to four times as long as for the normal child of his age. But he will learn to read and write a few words, count up to 10, add and subtract up to five, tell his name and address, age and sex, name objects and action in pictures, describe the use of common articles, understand daily routine, and obey simple commands. By adolescence, he can feed, care for, dress and undress himself, and perform routine chores, but he is incapable of managing himself or his affairs. His physique and personality are determined by the type of amentia. If it is inherent, his appearance is marred by stigmas of degeneration, paralysis, or epilepsy; if acquired, his being is comely, agreeable, and abiding.

Nontrainable, Noneducable Aments (Intelligence Quotient Less Than 25). The infant idiot is at his best in the first months of life, but heredofamilial degeneration of the nervous system soon produces mental retardation and disintegration. He may undergo slow motor development but rarely achieves the 1-year level in 4 years. He may learn to stand with support by the end of the third year but will not walk independently until he is 6. His neuromotor capacity is so feeble that he cannot eat solid food or learn to manage a spoon until the fourth year. He is conditionable within narrow limits, associating the bib and bottle, clothing and out-of-doors.

The idiot infant is apathetic but regarded as a very good baby during the first months of life. He eats and sleeps with regularity, gains weight steadily, and offers minimal difficulty in early rearing. He keeps his eyes wide open and his eye movements coordinated, giving a false impression of attentiveness. The mental deficiency may not be recognized until the child fails to sit up before the end of the first year. Soon enough the excitable infant becomes troublesome and destructive with his rhythmic movements, head-banging, head-rolling, grunting, snorting, tongue-chewing, scratching, brushing, body-swaying, and other stereotyped activities, which are fixations of patterns, not perversions.

The idiot child has the lowest type of mental defect with social incapacity, and is more helpless than the normal infant. He is usually incapable of doing anything for himself and must be washed and tended throughout life like an infant. He can be trained in some habits of cleanliness but seldom to the degree of a clever animal. He is able to speak crudely, walk sluggishly, and perform other muscular movements uncoordinately. He is mischievous, ill-tempered, and difficult to manage. He has a total incapacity for self-protection due to lack of understanding that such things as fire, traffic, deep water, and sharp instruments may be dangerous. The defect of mind may be so great that his instinct of self-preservation is completely absent. Indeed, he would die of starvation amid abundance of food if it were not put into his mouth. His imperfect physical development may give him a dwarfed, deformed appearance, and he may be afflicted with paralysis or epilepsy and die early in life from brain defect or intercurrent disease.

BEHAVIOR DISORDERS IN MENTAL DEFICIENCY

Cerebral Dysfunction. Mental deficiency is characterized not only by lack of endow-

ment but by lack of complexity of mind in both conation and affect. Instinctual weakness is manifest in utero by sluggish, feeble movements and after birth by ineffective sucking, slow feeding, and retarded development. Most infants tend to be unnaturally good and quiet, while some are repetitively overactive despite weak instincts and feeble feelings. Most retarded infants require one to four times longer to achieve developmental stages than normal infants although the step-by-step sequence of developmental events parallels that of normal children. Some infant aments, however, show overlapping in developmental tempo, i.e., motor skill appears relatively faster than language ability.

The entire developmental course is far behind normal as reflected by the delay in holding the head up, sitting up, prehension, standing, and walking. The limitation of experiential range of the ament's environment and the lack of initiative make him cling to infantile relationships with his mother until the fifth year. With little satisfaction and much frustration, the instinctual drives continue unabated in search of unrewarding activities that precipitate behavior disorders inappropriate for any occasion, i.e., head-banging, finger-sucking, air-swallowing, biting, chewing, dirt-eating, and body manipulation. The ament may learn everyday routine and conform to adult will, but he is unable to control the feelings raised by frustrations and explodes into panic or rage. This unpredictable behavior of the unstable ament led to the development and maintenance of state institutions.

The behavior difficulties of an ament are both intrinsic and extrinsic. Tredgold and Soddy[80] observed a deficiency in instinctual energy for adequate development, in native capacity for applying instinctual drives, and in innate control of impulses. The instinctual drives are directed toward objectives appropriate for a younger child, but continual frustration and abnormal stress accompanying these activities increase aggression and diminish self-control. The ament reacts not to the real issue but to his own imbalance between the drives and the situations. Extrinsic difficulties abound in the environment where the ament's individual needs are imperfectly understood but are minimal in an environment where his real needs are ministered to. The deficient or defective mentality does not deprive the child of his emotional needs. They are the same in kind but not in degree as those of the normal child. An ament's emotional adjustment is as individual as that of the normal child. Positive adjustment raises the mental level of physical functioning just as negative emotional adjustment lowers it. Kugelmass[79] demonstrated that treatment, training, and education must, therefore, be planned in terms of individual capacities of the child as a whole rather than in terms of the mental deficiency per se.

Evaluation of the ament usually reveals the underlying basis of the mental retardation. It may be due to endogenous deficiency in mental potential arising from genetic defect or to exogenous block in the reception of stimuli or in the expression of intelligence, despite normal mental potential. The former amentias, about 70 per cent due to inheritance, are endogenous, while the latter, about 30 per cent due to environment, are for the most part exogenous. Whatever the cause, amentia becomes manifest as a pattern of reaction to the child's environment.

Brain Damage. Localized brain lesions affect cognitive functions to a greater degree than conative and affective expressions. The instinctual drives are stronger

and more differentiated, better controlled, and more effectively modified. The aments with defects produced by exogenous causes have more energy and appear capable of more directional behavior than those with endogenous amentia. For example, in mild spastic palsy with a lesion mainly in the motor area of the cortex, there may be no cognitive defect but the mild ament may be so handicapped motorwise that his learning falls severely behind his intellectual capacity. The child attempts various objectives, becomes frustrated, and develops behavior disorders. Extrapyramidal involvement produces mental qualities comparable to the physical deviations. The ament shows a general rigidity of thought, a slowness in working out ideas, and difficulty in forming associations paralleling the general rigidity of movement and gross inability to adapt to changing situations.

Intracranial lesions produce mental retardation and loss of faculties; mid-brain lesions interfere with emotional control and expression. Choreoathetosis induces continuous involuntary and tortuous voluntary movements, while cretinism causes extreme mental and physical retardation with marked diminution in instinctual drives. Mongolism, emerging at about the third month of intrauterine life, diminishes the essential functions of mind. Postencephalitic syndromes are accompanied by overactive behavior as well as by parkinsonism. There is reduced capacity for coordinated movement and a mixed pattern of rigidity and defective emotional control. The child is usually explosive, hostile, unreliable, unaffectionate, and rejects all forms of training.

The origins of amentia accompanying developmental defects remain obscure for neither genetic nor environmental factors appear implicated in all aments. Even brain injury is not always the causative factor in these diverse phenomena, for many aments with developmental defects show no evidence of gross or minimal, diffuse or local brain injury. Fetal anoxia is a more determining factor in amentia accompanying developmental defect than postnatal anoxia. Oxygen reduction may occur in 15 per cent of the fetuses and subsequent difficulty in establishing respiration in as many as 10 per cent of the newborn infants. Anoxia definitely affects the brain and probably accounts for the diverse behavior disorders observed in aments with developmental defects.

Many studies of mental defect, however, fail to show significant differences between anoxic and control groups. Anoxia is usually evaluated in terms of the oxygen content of the blood, but the natal oxygen level shows poor correlation with subsequent intelligence levels. Blood oxygen saturation changes too rapidly to be of any significance in long-range prognosis. Nevertheless, intrauterine anoxia may well be presumed in the presence of maternal complications during pregnancy and labor with clinical signs of fetal distress. The slightest reduction in the oxygen supply during fetal life is deleterious to the infant. Yet Graham et al[21] observed that a large number of infants with anoxia show no cerebral impairment because of unknown protective factors that insulate them against brain damage.

Natal anoxia definitely causes subcortical brain damage responsible for overactive behavior. Ingraham and Matson[26] found cortical lesions involving the temporal lobes in aments with hemiplegia, aphasia, and homonymous visual field defects, and visual and gustatory auras in epilepsy. Gastaut et al[18] demonstrated electro-

encephalographic foci in the region of the temporal lobes, the unilateral temporal obe lesions giving rise to widespread dysrhythmia over the surface of the brain. Courville[12] correlated natal or postnatal anoxia with localized areas of cortical necrosis and scars, and particularly with striatal damage. Experimentally induced lesions of the temporal lobes produce overactive behavior similar to that observed in the series of aments under observation. Kluver et al[28] produced overactive behavior in monkeys after bilateral temporal lobotomy involving the uncus or hippocampal gyrus. The animals approached animate and inanimate objects previously feared and showed an irresistible impulse to mouth everything in sight and other behaviorisms paralleling those of aments.

The behavior of an ament with brain damage is the vectorial resultant of intrinsic injury and extrinsic defense patterns and environmental forces, with consequent retention of primitive patterns and delay in acquisition of new functions. The ament may be hypokinetic, inhibited in thought and action with attention fixed on one stimulus and activity limited to one motor discharge pattern and may remain withdrawn emotionally and physically. On the other hand, the hyperkinetic child is restless, irritable, grasping, investigative, jumping about from place to place. In the early years he combines his explorations with oral activity, while later he is brain-driven, darting about with choreiform or myoclonic movements. He finds difficulty with the control of impulses flooding the motor neuron but his outgoing activities are rarely satisfying. He has difficulty in perception and in visual, auditory, tactile, olfactory, and kinesthetic experiences. He is in a constant state of alertness, reacting to minor frustration with temper tantrums and bitter cries. He is driven by motor impulses beyond his control, pulled by fluctuation in muscle tone, handicapped by motor inadequacy and disturbed perception. He compensates for his difficulty by attempting to control his environment with incessant demands or becomes completely withdrawn and develops asocial behavior.

ORGANIC THERAPY OF INTELLECTUAL IMPAIRMENT

Psychopharmacotherapy. Specific types of mental retardation will be eliminated from developmental nonmaturation by special measures devised for the prevention of prenatal, natal, and postnatal causes of amentia. Some inborn errors of metabolism producing amentia can now be reversed by nutritional therapy. Phenylpyruvic oligophrenia in blond, blue-eyed, allergic children is due to an enzymic defect in the liver, which interferes with the conversion of phenylaline to tyrosine. Early recognition of this disorder in infancy by testing with 5 per cent ferric chloride added to an acidified urine enables maintenance of the affected infant on a low phenylalanine diet before amentia becomes irreversible. Galactosemia is another inborn error of metabolism involving an enzymic defect in the metabolism of lactose, which produces amentia, cataracts, malnutrition, jaundice, albuminuria, and galactosuria. Early recognition of galactose in the urine enables administration of galactose-free diets before irreversible brain and liver damage develops. Craniostenosis, recognized clinically and radiographically in early infancy, requires prompt craniectomy to insert the polyethylene film for relieving cerebral compression.

Nonspecific types of amentia require individualized symptomatic treatment in order to attain optimal mental, physical, emotional, and social development with minimal conflict in individual adaptation mechanisms. Parents are encouraged to use their own ingenuity in stimulating the child to self-care and self-reliance in the everyday routines, while aments are taught to be participants rather than spectators in order to acquire life habits essential for maintenance. The procedures will take several times as long as with a normal child, depending on the degree of retardation. About 75 per cent of child aments have an intelligence quotient of 50 to 75 and, therefore, are trainable and educable; 20 per cent with a quotient of 25 to 50 are trainable; while 5 per cent with a quotient less than 25 require custodial care. Recent advances in pharmacotherapy of mental disorders have raised hope that some compounds may be synthesized to stimulate neuronic growth or improve mental performance in mentally deficient children. Mental development is the vectorial resultant of spontaneous growth of the brain and of its stimulation by afferent impressions. Both forces work in unison throughout childhood, but the ament lacks the former and requires individualized training for the latter. Whatever drug, hormone, enzyme, or vitamin therapy might be developed, it would serve either to stimulate mental growth in reversible amentia or to develop the child's sensorium for improved training and accelerated learning.

Armchair philosophers deny the pathophysiological basis of pharmacotherapy in amentia of any type or degree. Every new experiment in medicine and surgery is considered inapplicable in the treatment of amentia. But clinicians in the field who select procedures applicable to individual cases and observe favorable results through the years of ament maturation have more hope for the social future of trained aments. Some pharmacological agents have a stimulating effect on cerebral metabolism, others have a relaxing effect, while still others alleviate systemic disorders that interfere with mental growth or performance. The study of new drugs presents many difficulties in the determination of dosage and effectiveness in comparative controls or double-blind studies. Beecher[6] observed that even the placebo produces a beneficial effect if accompanied by enthusiasm for the medication or encouragement by the physician.

Stimulant Therapy. Desiccated thyroid exercises striking physiological and pharmacological effects. Thyroxin or thyroglobulin has no special advantage for increasing mental acuity, overcoming mental obtundity, or correcting psychomotor retardation in the ament with substandard metabolic rate. Ross[56] observed an acceleration of cortical alpha rhythm and Cohen[11] noted an increased rate of oxidation of brain tissue with the rise in cerebral metabolism. I observed that the intelligence quotients of young hypothyroid aments increased during the first year of thyroid treatment, the mental levels remaining elevated on maintenance dosages of thyroid. Optimal dosage is estimated from height, chronological age, and bone age. The dosage is adapted to the individual and increased from time to time by 0.25 Gm. increments until the optimal therapeutic effect has been attained without exceeding tolerance. Benda[7] observed that calf whole pituitary powder is an effective adjuvant of thyroid therapy in the mongoloid infant with manifestations of hypothyroidism. Excessive therapy, revealed by excitability, nervousness, hyperactivity, irritability,

behavior disorders, tachycardia, sweating, and weight loss, calls for diminution or elimination of thyroid or, preferably, administration of a single dose once a week. The euthyroid ament tolerates thyroid better than the hypothyroid ament but tends to develop susceptibility to infection, nervousness, emotional instability, and sleeplessness. Discontinuance of the thyroid for a week with reinstitution at a lower dosage eliminates untoward manifestations.

Glutamic acid is a nonessential amino acid abundant in an adequate diet. Nachmansohn et al[37] observed its role in the enzymic synthesis of acetylcholine in cerebral metabolism. Price et al[40] found that the compound increased mental power. Zimmerman and Ross[51] confirmed this property by increasing the learning ability of white rats with glutamic acid. Albert et al[1] demonstrated a rise in intelligence quotient in 7 of 8 mentally defective persons. Later Zimmerman et al[52] recorded an average rise of 10.8 in intelligence quotient during the first six months, and less thereafter, in 30 aments with intelligence quotients exceeding 50. The best results were obtained with children devoid of organic brain damage. Quinn and Durling[41] found that glutamic acid stimulated mental and physical activity, and Zimmerman[57] noted the permanence of its effect in selected young aments. However, Milliken[34] failed to support this view; McColloch[36] found no mental improvement in any ament; and Heim[22] noted an intelligence quotient rise as the consequence of repeated testing by the same procedures. In my practice, I find the optimal effect of glutamic acid statistically significant in mild and moderate aments free from bizarre behavior. But in unselected cases, purposeless activity becomes greatly accelerated and the ament's behavior uncontrollable. In addition, recurrent acidosis during mild infections reflects an unstable acid-base mechanism disturbed by large dosages of glutamic acid.

Thiamine is the antineuritic vitamin indispensable in cerebral metabolism. Himwich[24] found subnormal oxygen utilization in malnourished aments. Since mental function depends on thiamine for oxidation of dextrose, the vitamin has been reinforced in the ament. Spies[46] observed striking changes in the electroencephalogram after thiamine therapy in frank deficiency. Others correlated disturbances of the higher nerve centers, i.e., forgetfulness, difficulty in orderly thinking, poor performance in school, vague fears, and irritability, with inadequate thiamine intake for normal tissue respiration. Rudolf[44] observed improvement in behavior in some aments and a rise in social quotients in some and in uncontrolled behavior in others. Thiamine stimulates neuronic activity but cannot supply intelligence or common sense. Excessive stimulation of lower neurons merely increases undesirable activity. I have never observed any mental or scholastic improvement on thiamine therapy except in the malnourished, institutionalized ament.

Amphetamine sulfate and dextroamphetamine sulfate give the disturbed child a feeling of well-being in a wide variety of behavior disorders, i.e., hyperkinetic, convulsive, nervous, schizoid, and postencephalitic. The drugs have a favorable effect on fear, depression, hyperactivity, and sexual tension and an unfavorable effect on severe amentia, schizophrenia, and psychopathic states. Amphetamine sulfate subdues the child but stimulates the adult. An ament becomes less active, less noisy, and less difficult. He shows quicker mental responses, greater attention span, better

cooperative effort. There may, however, be unfavorable effects from amphetamine sulfate, such as anorexia, irritability, seclusiveness, and sleeplessness. Administration of amphetamine sulfate in 20 mg. and dextroamphetamine sulfate in 10 mg. dosages daily is usually effective; if not, either compound may be gradually increased until a therapeutic effect is obtained. The daily quota is divided into two doses, given before breakfast and before noon. The resultant effect observed within one half hour is maintained for about five hours. Overdosage first causes restlessness, talkativeness, irritability, and insomnia and then confusion, delirium, convulsions, and coma.

Stimulants are invaluable for increased alertness, drive, and speech in the autistic child. Bradley (1950) found that amphetamine, 10 to 40 mg./day, and dextroamphetamine, 5 to 30 mg./day, controlled behavior disorders in children, especially restlessness, nervousness, hyperactivity, and distractibility. However, underactive and withdrawing children are stimulated by both compounds toward greater participation in childhood activities. Bender et al[8] observed favorable effects in neurotic and psychomotor behavior disorders but no improvement in psychopathic, schizophrenic, or brain-injured children. Ginn et al[20] considered dextroamphetamine valuable for controlling restlessness, short attention span, impulsiveness, and emotional instability in brain-injured children. Laufer et al[32] were able to increase photometrazol threshold to normal levels with dextroamphetamine. The threshold represents the specific light response evoked by a standard amount of pentylenetetrazol in mg./Kg., administered intravenously while simultaneously stimulating the patient by flashes of light from a powerful stroboscope at a set frequency. The end point is indicated by the appearance of a specific discharge in the electroencephalogram recorded as the "photometrazol threshold." A low threshold reflects diencephalic dysfunction. Iproniazid is equally effective in autistic children, increasing alertness in activity, awareness of surroundings, and drive for language. Stimulants may, however, produce excitement, liver dysfunction, and agranulocytosis.

Ataractic Therapy. The ataraxics include compounds derived from phenothiazine, *Rauwolfia*, diphenylmethane, and propanediol.

Phenothiazine derivatives studied by Heuyer et al[23] (chlorpromazine, 2 mg./Kg.) produced a calming effect in hyperkinetic, emotionally disturbed children. Freed et al[15] found that chlorpromazine, 10 to 250 mg./day, diminished hyperactivity in 72 per cent of the aments studied and improved learning capacity in 10 per cent of the cases, bringing about greater self-acceptance, closer interpersonal relationship, manifested by strengthening of emotional control, and better reading ability, but no improvement in intellectual functioning. Ayd[2] favored chlorpromazine for severe anxiety of schizophrenic children but observed occasional aggravation in the brain-injured children and in those with obsessive-compulsive problems. Gatski[19] observed that chlorpromazine, 40 to 80 mg./day, was effective in combating aggressive, destructive, hostile, and hyperactive manifestations.

Kugelmass[30] observed definite improvement in the behavior spectrum of mild and moderate aments, and Barr et al[3] demonstrated a rise in the intelligence quotient of some mentally retarded children. Rees[42] confirmed the improvement in behavior on chlorpromazine therapy, 30 to 75 mg./day, without any change in the underlying

psychopathology in schizophrenic children. Hunt et al,[25] however, found that chlorpromazine depressed disturbed children although it controlled hyperactivity and overcame isolationism. Denhoff et al[13] found chlorpromazine effective in the control of behavior disorders of children with cerebral palsy, but the drug did not improve muscle status. Freedman[16] observed the favorable effect of chlorpromazine on the behavior of schizophrenic children with respect to hyperactivity, anxiety, and interpersonal relationship. Other workers have given ample clinical evidence that promazine, 5 to 200 mg./day, perphenazine, 6 to 8 mg./day, trifluprομazine, 20 to 100 mg./day, promethazine, 40 to 100 mg./day, and prochlorperazine, 15 to 30 mg./day, yield results similar to those obtained with chlorpromazine with less toxicity in mild or moderate behavior problems but not in intractable cases.

Reserpine alkaloids diminish nervousness, irritability, and intractable behavior in children. Reserpine, 0.1 to 0.3 mg./day, was found by Talbot[47] to ameliorate irritability, hyperactivity, and sleeplessness in infants. Timberlake et al[49] determined by double-blind technique that reserpine, 1 mg./day, improved abusiveness, destructiveness, impulsiveness, and seclusiveness in less than half the cases in mentally retarded children. Lambros[31] used reserpine, 0.25 to 1.25 mg./day, to control hyperkinesia and enuresis. Zimmerman et al[53] supplemented anticonvulsants with reserpine to diminish petit mal, grand mal, and psychomotor seizures. Schwade et al[45] administered reserpine to improve severe behavior disorders of impulsive children with abnormal brain wave tracings. Lehman et al[33] observed some improvement in the behavior of autistic children but observed enuresis with large dosages of reserpine. Nicolaou et al[38] considered reserpine effective in the management of disturbed adolescents by altering psychological defenses and paving the way for corrective emotional experiences. But, Bender et al[9] found reserpine unpredictable yet helpful if the impulsive aggressive child with marked anxiety did not develop increased autonomic lability.

Diphenylmethane derivatives have a laudable effect on childhood emotionality. Bender et al[9] concluded that azacyclonol decreased impulsivity, integrated function, and reduced anxiety in both schizophrenic and brain-injured children. Bayart[4] observed a diminution of nervousness and psychic tension without sedative effects in disturbed children, leading to disappearance of tics within two weeks, of school difficulties within a month, and of emotional disorders within six weeks on continued therapy. Ayd[2] found that hydroxyzine, 75 to 300 mg./day, controlled the behavior of neurotic and hyperkinetic children by lessening tension, overcoming hyperactivity, and relieving disturbed autonomic function, but the results were not as striking as with chlorpromazine or reserpine. Freedman[16] considered hydroxyzine effective in the management of behavior disorders by reducing anxiety, overcoming nervousness, and improving sleep patterns. Diphenhydramine, 130 to 160 mg./day, was found by Effron et al[14] to be effective in primary behavior disorders. Bender et al[9] used diphenhydramine to relieve impulsive behavior in disturbed children, especially head-banging, tics, and other motor activities.

Propanediol derivatives produce favorable effects in organic brain disorders. Perlstein[39] demonstrated the effectiveness of meprobamate in convulsive disorders, especially petit mal, as well as in serious behavior disorders. Litchfield[34] observed

positive results with dosages ranging from 50 to 800 mg. in prepsychotic children. Livingston[38] considered meprobamate, 400 to 2400 mg. daily, to be more effective than chlorpromazine or reserpine in the management of behavior disorders in epileptic children. But, Kugelmass[39] found meprobamate most effective in the management of children with behavior disorders with and without convulsive manifestations.

Diphenylhydantoin sodium is effective in behavior disorders associated with electroencephalographic changes resembling epilepsy, especially irritability, hypermotility, and variability of behavior. It is a stimulant rather than a sedative drug. A young child requires 0.5 Gm. three times daily, mixed with cream to disguise the bitter taste and to diminish gastrointestinal irritation. The older child requires about 1.5 Gm. three times a day, given in capsule form. Diphenylhydantoin sodium does not have the hypnotic properties of phenobarbiturates. Hyperplastic reactions of the gingivae may be overcome by ascorbic acid, but other toxic manifestations require discontinuance of the drug. Paraldehyde has a calming effect on an ament in a state of high excitement. It may be given rectally, 1 teaspoonful in 1 oz. of oil by retention enema, or intravenously, if necessary.

Mephenesin is a muscle relaxant in spastic disorders involving the region of the thalamus, internal capsule, cortical nuclei, or brain stem. It provides a state of relaxation with a pleasant feeling of warmth, numbness, and euphoria for two to three hours. Muscle spasm, spasticity, rigidity, tremor, and startle often disappear and the child is able to fall asleep normally during the effective period. The usual dosage ranges from 0.75 to 1.5 Gm. daily.

Dibenamine blocks excitatory sympathetic responses without disturbing the inhibitory effects. It is a synthetic nitrogen mustard derivative too costly to be administered by any other method than dilute intravenous drip of 2 to 3 mg./Kg. in 500 cc. saline in one hour. Unfortunately, the number of side reactions, such as mental confusion, visual impairment, nausea and vomiting, and loss of bladder control, limit its usefulness. Insulin in subshock dosage is produced by hypodermic injection of regular insulin beginning with 5 units daily and increasing by units until the precomatose stage of classic insulin therapy is attained. After about 30 units, the child manifests hunger followed by fall in body temperature, salivation, and increased perspiration. The injections are given daily for two or three weeks and the insulin dosage gradually reduced.

Toxicity. Potent psychochemotherapeutic drugs reveal potential toxicity. Phenothiazine derivatives may produce agranulocytosis, jaundice, swallowing difficulties, parkinsonism, dermatitis, hypotension, sinus tachycardia, convulsions, urinary retention, and somnolence. Reserpine derivatives may produce leukopenia, convulsions, parkinsonism, depression, and hypotension. But diphenylmethane derivatives produce few toxic manifestations, although drowsiness and leukopenia may be transient and reversible. Propanediol derivatives may produce purpura, diplopia, dermatitis, hypotension, diarrhea, and convulsions as withdrawal symptoms.

CLINICAL ASSESSMENT OF PHENAGLYCODOL THERAPY

Phenaglycodol Studies. Psychopharmacotherapy has a rational role in the limited armamentarium for the management of mentally retarded children. A national survey

of the current opinions and clinical experiences of leading investigators in the field of amentia regarding the applicability of the newer ataractic agents revealed considerable diversity of opinion regarding the indications of ataraxics, choice of drug, mode of application, and duration of effectiveness. My experience[29] with a group of carefully followed mentally retarded children given intensive treatment with ataraxics provides ample support for such therapy. The superiority of meprobamate in comparison with other compounds as an adjuvant in the symptomatic therapy of ament disorders led to the clinical evaluation of phenaglycodol in mentally deficient children. Sixty moderately retarded aments 3 to 18 years of age, with an intelligence quotient range of 30 to 75, were maintained on phenaglycodol in gradually increasing dosages to determine its effect on the specific manifestations of unmanageable behavior.

The symptoms exhibited were remarkably constant in the series of aments studied, more marked in the younger than in the older group. There was overeating, messy feeding, and gagging at meals. The constant noisiness, utter destructiveness, and unpredictable violence perpetrated by these aments were not amenable to reprimand or punishment for more than a minute or two, if at all. Even the children who knew what was considered right or wrong failed to resist the compulsive impulse to touch, mouth, or tear objects or interfere with others about them. The hyperactive children were invariably restless, tireless, and fearless, with temper tantrums in the unstable and convulsions in the epileptics. During aggressive phases, the children would bite, hit, and kick everything and everyone in sight. The high energy potential for overactive behavior precipitated marked distractibility, total inattentiveness, and fluctuating moodiness, mental attitudes not conducive to training. About half the children remained enuretic and a third incontinent despite attempts at toilet-training.

The plan of the study consisted of a one-month pretest observation period before initiating the drugs, followed by a two-month period on the selected drugs, and finally a one-month period of discontinuance of all medication, except anticonvulsants. Half the group were offered phenaglycodol after meals and at bedtime and the other half the identical placebos. After the two-month period, the phenaglycodol group were given the placebo and vice versa. The author alone was aware of the type of drug offered the children. The dosages determined individually for each child ranged from 400 to 2400 mg./day according to the severity of the behavior disorders. Side effects in 4 children at the onset of the study required elimination of 1 from the group and diminution of the dosage in the other 3; 4 additional aments were selected to replace these equivocal cases eliminated from the study.

All groups of mentally retarded children maintained on phenaglycodol or placebo showed statistically significant increments of improvement in several behavior categories according to the chi-square test. Preliminary orientation with the drug indicated progressive improvement for four to eight weeks and, conversely, progressive regression on withdrawal of the drug within two weeks, ranges found statistically significant with the chi-square test. The patterns of deviant behavior were as unique as the individual ament. They reflected the child's mental "garment" and appeared amenable to alteration. The absence of valid measurements for such individualistic

TABLE II

Differences Between Phenaglycodol and Placebo Effects on Behavior Disorders in Child Aments (Number of Patients out of 60 Revealing Positive or Negative Changes, Omitting the Number Showing No Significant Behavior Changes or Side Effects)

Behavior difficulty	Phenaglycodol		Placebo	
	Improvement	Worsening	Improvement	Worsening
Feeding: gagging, messiness, overeating	26	12	2	3
Dressing: resistive, uncooperative, untrainable	22	8	1	2
Compulsion: touching, mouthing, smelling	24	8	6	8
Control: noisiness, nervousness, fidgetiness	32	11	8	5
Activity: restlessness, aggressiveness, destructiveness	30	10	3	2
Attacks: tremors, temper tantrums, convulsions	34	14	3	4
Training: distractibility, inattentiveness, moodiness	28	6	2	1
Toileting: enuresis, incontinence, messiness	20	12	2	2
Bathing: resistive, uncooperative, untrainable	25	5	4	7
Sleeping: sleeplessness, sleepwalking, night terrors	25	4	5	2

behavior led to grouping of the irreproachable conduct into 10 special categories (table II).

Some of the children selected for this study lived at home; hence the reliability coefficient of the 36 pairs of ratings was 0.78. Most of the children lived in special schools; thus the reliability coefficient rose to 0.85 with more refined techniques in obtaining objective nonevaluative descriptions of the changes effected by the drugs.

TABLE III

Phenaglycodol Effects According to Age

Behavior deviation	Age, yr.					
	1-6		6-12		12-18	
	+	−	+	−	+	−
Feeding	4	2	10	4	12	6
Dressing	2	1	8	3	12	4
Compulsion	2	1	9	3	13	4
Control	3	1	12	3	17	7
Activity	6	2	8	2	16	6
Attacks	5	2	12	5	17	7
Training	2	1	10	2	14	3
Bathing	2	1	8	2	15	2
Sleeping	2	1	9	2	14	1

TABLE IV

Intelligence Quotient Levels of the Children According to Age

Intelligence quotient	Age, yr.		
	1–6	6–12	12–18
<25	2	3	4
25–50	6	14	12
50–75	2	7	10

Both parents and attendants were trained in objective reporting of ament behavior and basic skills before commencing the experimental observations. The obvious weaknesses of such multiple rating techniques were compensated to some extent by comparative studies of the two groups and by the weekly evaluation of each of the children by the author. We assessed the degree of disruption of daily routines in 10 groups of skills since mental retardation interferes with the gradual acquisition of these functional performances in the course of early development. The 10 areas of behavior studied represent the composite of a child's overt but not total behavior, yet the results reflect significant changes in such behavior during the experiment. The psychological effect of the new treatment was overcome partly by the administration of a placebo, partly by prolongation of the experiment, and partly by the analysis of variance.

Most of the aments showed little or no change in individual behavior except for mild side effects until relatively large dosages of phenaglycodol were administered toward the end of the initial experiment and during the final period of therapy. There was a significant difference between the mean ratings of child aments maintained on phenaglycodol and on placebo, a far greater number showing definite improvement in comparison with those who showed worsening on the drug therapy during the experimental periods. Individual analysis of the children who improved revealed distinguishing characteristics of such improvement and thus established the graded criteria of differential changes in predicting positive effects of phenaglycodol therapy. Indeed, there was a beneficial effect of the drug therapy sensed in both home and school throughout the period of study. The mean ratings of the placebo groups in each area of behavior were limited to an insignificant number of child aments who showed improvement or worsening, since most of the children revealed no change in behavior on placebo therapy.

TABLE V

Phenaglycodol Effects According to Type of Amentia

Type	Age, yr.					
	1–6		6–12		12–18	
	+	–	+	–	+	–
Organic	3	1	8	2	9	3
Functional	5	1	11	3	12	2

TABLE VI

Phenaglycodol Side Effects (Number of Cases out of 60)

Minor		Major	
Drowsiness	2	Angioedema	1
Irritability	1	Hyperactivity	1
Lethargy	1	Leukopenia	1
Unsteadiness	1	Urticaria	2

The effectiveness of phenaglycodol in managing the disturbed, mentally deficient children was adjudged on the basis of degree of alteration of the 10 groups of behavior patterns, the results being grouped in terms of degree of improvement or worsening apart from side effects, as given in table II. The home and school groups of aments under study underwent favorable changes in personality as a whole and in specific areas of behavior. The hypersensitive became quiet; the unmanageable, cooperative; the rebellious, responsive. However, a fair portion of each group showed little or no change and often some worsening of the manifestations comparable to that observed with barbiturate therapy. The children with functional behavior disorders responded better than those with deep-seated organic defects. The personnel in the schools and in patient's homes were gradually released from the time-consuming, enervating responsibilities of severe discipline and physical restraint to devote themselves to the individualized training and recreation of the mentally retarded children.

The results of the combined studies of 60 mentally retarded children indicate 43 per cent improvement and 20 per cent worsening with respect to gagging, messiness, and overeating as difficult feeding problems; 36 per cent improvement and 13 per cent worsening with respect to resistant, uncooperative, and untrainable reactions to dressing and undressing routine; 40 per cent improvement and 13 per cent worsening with respect to touching, mouthing, and smelling compulsions; 53 per cent improvement and 18 per cent worsening with respect to noisiness, nervousness, and fidgetiness as manifestations of poor body control; 50 per cent improvement and 16 per cent worsening with respect to restlessness, aggressiveness, and destructiveness as uncontrollable activity; 56 per cent improvement and 23 per cent worsening with respect to tremors, temper tantrums, and convulsions as manifestations of easily provoked attacks; 46 per cent improvement and 10 per cent worsening with respect to distractibility, unattentiveness, and moodiness as deterrents of everyday training; 33 per cent improvement and 20 per cent worsening with respect to enuresis, incontinence, and messiness as manifestations of bad toilet habits; 41 per cent improvement and 8 per cent worsening with respect to resistive, uncooperative, and untrainable responses to bathing routine; 41 per cent improvement and 6 per cent worsening with respect to sleeplessness, sleepwalking, and night terrors. There was no significant difference between the test and retest results on the Terman-Merrill scale of the phenaglycodol-treated children. The pharmacological effect of phenaglycodol involved qualitative alteration of the psychological defenses that allay anxiety. The compound cannot correct the deficient capacities in the child but apparently

does enable the available capacities to function more effectively under individualized or group training during optimal phenaglycodol therapy. The drug must be continued uninterruptedly; in our study most of the positive effects disappeared after discontinuance of the drug, most of the treated aments returning to their incorrigible behavior within a fortnight.

CONCLUSIONS

1. Mental deficiency is evaluated in terms of underlying systemic disorders and classified according to psychometric tests.

2. Behavior disorders in mental deficiency are viewed in terms of cerebral dysfunction and interpreted according to the type of brain damage.

3. Organic therapy of intellectual impairment is discussed in terms of stimulant and ataractic compounds affecting deep-seated brain centers.

4. Clinical assessment of 60 mentally retarded children who were adequately controlled on phenaglycodol therapy revealed statistically significant improvement in 10 fundamental areas of behavior according to the chi-square test, i.e., in feeding, dressing, compulsion, control, activity, attacks, training, toileting, bathing, and sleeping.

5. Phenaglycodol enables the available but delimited capacities of the mentally retarded child to function more effectively, without side effects, throughout the period of administration.

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Childhood Schizophrenia, Childhood Autism, and Heller's Disease*

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The child psychiatrist who is faced with the difficult problem of differentiating the various conditions leading to abnormal behavior patterns in children is confronted with a number of conditions, all of which may result—at least temporarily—in similar mental symptoms. During the last century and at the beginning of the present one, psychiatrists were mainly concerned with adult patients and very little was known about children, except that some of them were mentally defective or were what used to be called "idiots."

Dealing with insanity in adult patients, who usually had been normal until the late teens, Kraepelin[1] developed the concept of dementia praecox as a process psychosis, which would gradually lead to complete mental deterioration and complete dementia. Bleuler[2] enlarged the morbid entity of dementia praecox by introducing the concept of schizophrenias, including various forms of schizophrenic behavior patterns, and by showing that by no means every development resulted in a state of deterioration. It is notable that both great psychiatrists were rather slow in accepting the idea that schizophrenia could develop at a much earlier age and even early in childhood.

Parallel to the concept of childhood schizophrenia arose the concepts of dementia infantilis or Heller's disease and childhood autism. All these conditions have to be differentiated from the chronic brain syndromes in children that are residues of developmental disorders and so-called "birth injuries," mainly asphyxiation at birth and progressive degenerative diseases of a metabolic nature, the lipoidoses and leukencephalopathies. Even the early workers in the field of mental deficiency realized that there was one form of idiocy, often occurring in rather beautiful children, in which the child acted "idiotic" and yet maintained a pleasant and graceful appearance, while exhibiting strange behavior patterns, which often revealed flashes of adequate intelligence. The autopsies of these children disclosed so-called "normal," well-developed brains with no pathology demonstrable by methods available at that time, thus leading to the concept of idiocy with a normal central nervous system.

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In order to differentiate and analyze the conditions of childhood autism, Heller's disease, and childhood schizophrenia, some preliminary remarks are necessary since many authors use the terms almost interchangeably and hence no clear lines of demarcation are available. The best definition of childhood autism has been given by Leo Kanner,[3–5] to whom we owe the description of the syndrome:

> The common denominator in all these patients is their disability to relate themselves in the ordinary way to people and situations from the beginning of life. Their parents referred to them as always having been "self-sufficient," "like in a shell," "happiest when left alone," "acting as if people weren't there," "giving the impression of silent wisdom." The case histories indicate invariably the presence from the start of *extreme autistic aloneness* which, wherever possible, disregards, ignores, shuts out anything that comes to the child from the outside. Almost every mother recalled her astonishment at the child's failure to assume at any time the usual anticipatory posture preparatory to being picked up. According to Gesell this kind of adjustment occurs universally at 4 months of age.

Many of these children are found to be mute and refuse to talk at all except in single words, which they usually utter in a rather explosive way. It is comforting to know, however, that of 20 children of Kanner's[3–5] group, 13 acquired the ability to speak while 7 remained mute. He stated, "But language, even when present, did not over a period of years serve to convey meaning to others. Naming presented no difficulty; even long and unusual words were retained with remarkable facility."

Kanner[3–5] described childhood autism well as a syndrome of a particular nature. He probably felt that childhood autism is an early manifestation of childhood schizophrenia and that most autistic children will remain schizophrenic after puberty. Few children are able to evolve from the stage of autism and make a more or less adequate community adjustment.

We feel that Kanner[3–5] made a great contribution in coining the term "childhood autism" but also feel that while it clearly describes certain behavior patterns in children, childhood autism should not be identified with childhood schizophrenia. Autism may be due to asphyxiation at birth, to severe emotional deprivation in infancy and early childhood, or to metabolic disorders, as well as to a beginning schizophrenic process. In using the term as a very adequate description of specific behavior, one should not be prejudiced as to the classification of the child's behavior in one of these categories.

As for dementia infantilis, the most adequate description of the condition was provided in 1930 by Theodore Heller:*[6]

> A number of cases among an extraordinarily large number of mentally defective children attracted my attention in 1905 and 1906. The histories were so similar that there was no doubt that the cases belonged in a category of their own. The children came from different countries and belonged in different social strata. The children were in their third and fourth years of life. Without previous illness, a change in character could be noticed which was especially evidenced by a change of mood. Formerly quiet and vivid, they became irritable, negativistic, disobedient. They had unprovoked outbursts of temper, cried and destroyed toys with which they had previously played normally. Many showed anxiety, sometimes of

* Translation from the German.

a hallucinatory character, and with the occasionally insidious increase of violent manifestations, a mental regression took place which led to a complete loss of speech and complete deterioration within a few months. Language became poor with gradual transposition of words, the child was unable to repeat sentences and finally stopped speaking. Perception of speech was also lost except for a few primitive remnants. A change in motor behavior became manifest during this process of deterioration. The children developed automatic movements, tics or grimacing, and manifested certain odd positions which would remind one of the erethism of some idiots. Many of them had to be fed and became unclean and incontinent. A certain degree of tension was evidenced by the eye movements.

All parents without exception made the statement that the children had been intelligent before the onset of the illness, in some cases indicating that their children had been especially gifted. These statements had to be taken with some reservation, however, because it is well known that many parents overestimate the intellectual capacities of their children, especially in retrospect. Still there can be no doubt that the children had spoken, had been able to understand and had acted independently according to their age. These children had not been mentally retarded or especially conspicuous.

The intellectual loss usually came about within nine months, and no improvement had been noticed in any of the cases. The children remained in a condition of complete mental deterioration. Motor overactivity, stereotyped behavior and mannerisms with tics were most conspicuous. Many of the children retained their intelligent expression, especially the clear look and apparent attention with which they observed their surroundings. They were not fit for any mental training although some of them again learned to feed themselves and could be toilet trained.

The parents usually expected that the children would regain their lost mental capacities, and were deeply disappointed when the mental deterioration and low mental standing remained and the children deteriorated completely.

Various authors have described cases of dementia infantilis (Higier[7] and Jancke[8] and others), and in 1921 Julius Zappert[9] presented 13 cases, distinguishing stages observed in all cases as follows: (1) normal mental and physical development in the first years of life; (2) beginning of the illness between the third and fourth years; (3) changes in psychological and mental behavior in the way of failure of further education and rapport; conspicuous overactivity; irritable, frightened behavior; increasing mental deterioration; (4) difficulties in speech at the beginning and during the course of the illness; (5) preservation of motor function and absence of any focal symptoms of the central nervous system; (6) complete mental deterioration in the end stage; (7) intelligent expression of face and eyes.

To quote Heller[6] further:

I now have available 28 cases of dementia infantilis, all of which had been examined carefully by medical specialists so there is no confusion with other morbid entities. It seems especially important that no cases of encephalitis lethargica had been included, and no cases where the parents' statements were not reliable. In all cases, progress of the illness was exactly the same.

Especially interesting is the final fate of these children who all remained idiotic, did not talk, did not understand when spoken to, and were unable to occupy themselves. They were restless and manifested tics and mannerisms after many years, similar to those observed in the very beginning of the illness. Most of these children had been sent to state hospitals because it was impossible to manage them on account of their restlessness. Fever therapy with malaria was tried in one case, without success. In spite of their idiocy, many of these patients who had grown to adulthood gave a much more intelligent impression than other idiots with whom they shared the dormitory. The facial expression and look of the eyes

in many cases led the unhappy parents to hope that the patients would finally regain their mental capacities. The parents went from doctor to doctor, requesting therapy.

E. Redlich has dealt with the question of whether these cases are identical with early manifestations of dementia praecox. In the very beginning he felt that there was a certain similarity between dementia infantilis and some forms of schizophrenia but he finally came to the conclusion that dementia infantilis was a particular morbid entity which was not identical with the group of schizophrenias. The cause of dementia infantilis appears unknown.

Since the position of dementia infantilis among the morbid entities is still a matter of argument, the diagnosis is often disputable. It is only natural that the cause of Heller's[6] disease would be equally in question. There are very few autopsy reports in the literature. Vladimir Hudolin,[10] who recently reported a case and discussed the diagnostic problem, mentioned a case of Moyano, Outes, and Carillo[11] in which extensive atrophy of the frontal lobes was found. The brain weighed only 750 Gm. Several writers are of the opinion that the condition developed subsequent to encephalitis, and quite a number of authors consider dementia infantilis a postencephalitic condition. Another group of investigators favored a theory of a metabolic disorder. Leo Kanner[3–5] was inclined to adopt Corberi's theory[12] that this condition represents a "cerebral degenerative process akin to Tay-Sachs disease and to a similar process described by Kufs and by Walter as 'amaurotic idiocy without amaurosis.'" Paul Yakovlev[16] seemed to be leaning toward the same opinion.

In summary, four main theories are in vogue at present: (1) Heller's syndrome is a clinical syndrome not due to one etiological factor; (2) Heller's syndrome is symptomatic of a postencephalitic condition; (3) Heller's syndrome is related to childhood schizophrenia; (4) Heller's syndrome represents a form of progressive deterioration due to a metabolic disorder. A new autopsy report will be given later in this paper.

In studying childhood schizophrenia, we are faced with not only the special problems of childhood but also the problems encountered in an understanding of the psychotic process. In adult schizophrenia, we often deal with persons who seemed perfectly normal before they fell ill and have recovered from these conditions and are often able to give an excellent account of their experience. In childhood schizophrenia, we are actually concerned with three different groups, which should not be confused: (1) It has become more and more obvious that dementia praecox, as described by Kraepelin, often does not start after puberty, but that children in the prepuberty and early puberty periods may already give indications and develop a symptom complex that may justly be called a schizophrenic psychosis. In this group we have children who have been normal, occasionally highly intelligent up to 10 to 12 years of age, but then change in their attitudes and mental processes and reveal evidence of compulsive behavior patterns, depressive moods, or other symptoms including hallucinations, which are considered significant by many writers. (2) Some children fall ill in the 6-to-10-year age group, children who have previously learned to talk and interact with their environment. Thus again, we are looking at patients who had been considered more or less normal up to a certain time when a true change in personality took place. Although the actual diagnosis may often be difficult, we are on rather firm ground in a considerable

number of cases and apply the term "schizophrenia" with at least the same justification as its use by psychiatrists in regard to adults. (3) In the last group we deal with children who have never been "normal" except for their physical appearance and neurological complexes, so that any evidence of an organic brain syndrome can be excluded. These children seem promising in physical appearance and motor performance but reveal peculiar behavior patterns, which show lack of communication, lack of reality contact, and usually lack of speech as a main channel of interaction with their surroundings—in brief, the autistic child.

It is obvious that the syndrome of childhood schizophrenia encompasses a very large field of mental attitudes and abnormal behavior patterns. It is impossible at this point to discuss those forms of childhood schizophrenia that, as mentioned before, developed around puberty and early in the teens. We want to restrict our presentation to that group of cases where a child (early in life) shows an abnormal behavior pattern that cannot be properly classified. The situation is complicated by the fact that most of these children are so disturbed that their mental performance is subnormal for practical purposes and cannot be properly evaluated. However, there are a few children who may show even superior intelligence, and we would like to start our presentation with discussion of a child who has attracted some national attention.

In 1954, an article appeared in a periodical under the sensational title, "A little, lonely genius. An 8-year-old boy with an IQ rating of 185 takes 10th grade studies in his stride," and compared the child with Einstein, who is said to have had an intelligence quotient estimated at 192.

One of us (C. E. B.)[1] has known this child (B.) since 1953, when he was almost 8 years of age. His older brother, one year his senior, was an intelligent, "average" boy. When B. came to the office for a consultation, one could hear him outside talking for a long time in a very abrupt, loud manner. He was asking countless questions: What is this? What is that? and opening every door, entering every room possible, and riding up and down in the elevator, which fascinated him. He was completely unrestrained while Dr. B. talked with his parents, touching every object he could reach, examining everything, and keeping the whole environment under constant strain and tension.

B. was physically well developed, a handsome child with a somewhat large head. He would not take off his coat or cap and would not sit down for one moment but walked up and down and kept as far as possible away from the examiner. On first impression, there was no doubt that he had a childhood schizophrenia. However, when alone with Dr. B. he gradually became more responsive and cooperative and revealed an amazing knowledge and ability in definitions, reading, and verbal expression. He had brought a book of fairly advanced physics, from which he read fluently, and he was able to give many complicated definitions on the level of a high school boy of 16 years. He knew many words and terms far beyond his own age level, and in this line of performance scored an intelligence quotient rating of about 180.

In contrast to B.'s verbal abilities, he had a poor grasp of number facts and arithmetic problems, in which he showed no interest or willingness to concentrate, even for a fraction of a minute. There were many strange patterns in the boy's behavior. He was extremely restless, completely unrestrained, rather destructive, and raised havoc with everything he could get hold of. As mentioned, he would not sit down or take off his coat. He refused to write a single word or name for the interesting reason that by writing his name he would give away something of his personality.

It took three full years to get the boy to write his initials: B.V.D., and it was several months longer (when he was 11 years old) before he would write his name and address. He wrote in printing fashion, rather stiff and irregular and even with a slight tremor.

As far as the history is concerned, B. is said to have developed normally. He walked at 11 months but would not talk. It was not until he was 3 years old that he tried to talk, but when he finally decided to use language he is said to have picked up a newspaper and read it aloud to his parents. At the age of 4

years he is said to have read the baby sitter's high school biology textbook when he was supposed to be sleeping. The baby sitter was found to be completely unnerved because he bombarded her with endless questions about high school biology, which she was unable to answer. When he entered the first grade at the age of 6 years, B. ran around in the classroom, refused to sit down, and finally walked out with the authoritative statement: "I don't like what they teach here." Private tutoring was attempted, but the psychiatrists who were consulted at that time felt that the child was schizophrenic and would not maintain this intelligence quotient rating.

When seen at the age of 8 years, B. talked a great deal to himself and showed flight of ideas, lack of concentration, and complete lack of restraint. Emotionally he was more immature than the normal child his age, but intellectually he was far advanced.

In our opinion we have here one of the purest examples of accelerated mental development, in contrast to deceleration of mental development, which is such a common observation in children who are called "mentally retarded." We have always postulated that if we have decelerated mental development, we should also find accelerated forms of development in which the brain rapidly reaches a higher stage of maturation, even as premature sexual maturation is occasionally observed as an isolated finding. We do not think we are justified in making a diagnosis of schizophrenia and prefer to think of this child as a person with accelerated mental development in which, indeed, a sort of split of personality is present because the emotional maturation has not kept pace with the mental status. However, such an occurrence does not need to signify a mental illness.

It is always rather gratifying if one has an opportunity to follow up a child over a long period, and the following report is on a child whom one of us (C.E.B.) had an opportunity to see over a period of 10 years.

This boy (S.) was first seen in our outpatient clinic in 1948, at the age of 3 years, 9 months. He appeared well developed and well nourished but refused to talk or cooperate and screamed constantly at the top of his lungs. He tended to toe in when walking, and the knee and ankle jerks appeared very active, but there was no Babinski reflex. In the psychological examination, S. did not talk but uttered a few words when so disposed. He was entirely withdrawn and autistic and had not established any contact with the outside world. He seemed restive, aggressive, and destructive. It was evident that the boy would fail in any scholastic ability test because he refused to do things that a child of 2 years could do without difficulty. He was able to put on his stockings and looked for his shoes, would come sometimes when called, and was very anxious to guide the family from the examination room. It was evident that the child knew much more than his family realized. Our neuropsychiatric evaluation was as follows: "S. is a withdrawn child who would also be classified as an autistic child. He has developed good motor control and coordination and acts according to his own will. He refuses to cooperate or comply with requests from the outside. There is no doubt that S. represents a serious psychological problem, and we would advise that something be done about it. He needs special coaching in speech as well as psychological attention."

This child was re-examined two years later, in 1950. At that time he was a very well-developed boy who again was extremely fearful and uncooperative, screaming and only quieting down when he thought the examination was over and he could go home. He was not only uncooperative but violent and destructive, breaking two glass windows in a bookcase by kicking with his legs. In contrast to the 1948 examination, the boy spoke clearly in sentences. However, he repeated stereotyped phrases endlessly and all with a strange lack of emotional involvement. It was impossible to give him any rating. In the two years, he had made great progress in language and no longer appeared autistic, but he was now a thoroughly disturbed child with a great amount of anxiety, which was especially caused by the mother and an aunt, who hovered over him. Two examiners independently made the observation that these women hovered over the child "like two huge, strange birds," and scared the child, and sometimes S. seemed to come to the examiners as if looking for help and protection.

Now, eight years after the second examination, S. is $13\frac{1}{2}$ years old. He is a handsome boy and does not give the impression of being abnormal until one has had an opportunity to get in close contact with him. He attended the Roxbury Children's Center for several years but could not progress beyond a certain level. He is now placed in a special class, where he does first- to second-grade work. He prints his name in large letters and can spell a few words. He makes some simple drawings on a much lower age level and in general deserves the term "retarded." However, his behavior is extremely odd. He is full of anxieties and thoughts, constantly fearing that something will happen to him. He thinks other children will beat him up, and he repeats sentences in a rather monotonous, loud voice like a machine gun. He talks in a rather differentiated way—mentions that the other children torture him and throw him down, call him a mental case. The mother reports that S. goes out alone quite a lot, spends his days at playgrounds watching the other children but not participating. He goes to school alone and returns at the appointed hours.

Since special education in the community has not been successful, an attempt was made to admit S. to a school. There were objections to putting him in the children's unit, and I hoped that the boy might adjust to the neutral environment of a state school. I may remind you of a report by Dr. Rabinovich of two such autistic children with extremely low intelligence quotient ratings who were admitted to a state school under the diagnosis of "mental retardation" and gradually came up to ratings greater than 100. This observation seems to indicate that a child removed at the right time from an overbearing, unsuitable environment might quietly develop in an environment of a neutral nature where there is little stress. I have to confess, however that we managed the case very poorly. The boy, who had never been away from home, was not permitted visitors for a long time. On the first general visiting Sunday, when everybody talked about the visitors, other boys asked S. whether his mother would come, and he was sure that he would have visitors. Then another boy teased him and said: "No, your mother is dead," whereupon S. developed an extreme state of anxiety and became completely unmanageable. The physician finally decided to telephone the family and have them visit the child, but when they came they found S. in such extreme emotional anxiety, clinging to his mother, that they did not dare leave him and took him home. There were other incidences of inadequate behavior and actual disregard of rules on the part of physicians and attendants, demonstrating again how difficult it is to train an attendant staff of a state school to manage emotionally disturbed children in a suitable manner. The boy is now at home, having gone through a new, extremely unfavorable experience, which will make it even more difficult to separate him from his home and place him in adequate school conditions.

This second case, a child who learned to talk and report his experiences rather adequately, stands between the so-called "normal" child with mental disturbance and the disturbed child who never expresses himself in an articulate fashion so we never know what is going on in him. We have seen a number of children in the latter category, who never adjust or learn to make a community adjustment, and most of them eventually have to enter a state school under custodial care. There is no other place for them, and when they reach their teens, they do not fit into a mental hospital for adults.

A recent book on *Ego Structure in Paranoid Schizophrenia* by Luise J. Zucker[13] opened with the statement: "It is commonly accepted that the ego structure is weak in schizophrenic conditions." This fundamental statement points out one general aspect of schizophrenic behavior in the scientific language of ego psychology. One may well go along with this scientific formulation, and most modern writers will agree that the schizophrenic patient—child or adult—reveals a weakness in contact with his environment, that his ego strength is little consolidated and his ego boundaries fluctuating. One may even go further and accept the scientific equation that "the ego is exposed to urgent demands from three different sources: the outer reality, the id and the superego. It is thus caught between the intensity of the id

drives, the judgmental, condemning effect of the superego, and the impact of the external world."

It is also true that under different conditions, given boundaries are affected in a different manner. In hysteria, the boundary between the ego and the outside world is considered weak, the external experiences are absorbed and incorporated, and the ego boundary is so fluid that the individual participates too much in the lives of others, identifies too readily and too strongly. On the other hand, the boundary between the ego and the id is rigid and the ego is unable to accept its own drive nature. In contrast to this, in the obsessive-compulsive conditions, the relationship with the outside world is walled off, rigid, and unflexible. The person is unable to interact freely with the external world. On the other hand, the ego identifies readily with its own impulses and drives. In psychiatric conditions, both boundaries — the one toward the external world as well as the one toward the id — seem poorly established.

These ego models are useful to a certain extent, but should a diagnosis of schizophrenia be based solely on poorly established boundaries? And if not, what is the difference between the person with poor ego boundaries and the person suffering from a schizophrenic process? Are the so-called "secondary" symptoms of schizophrenia readily understood from a theory of weak boundaries, or have we still to postulate other factors that would make such a total personality collapse understandable?

For an understanding of early childhood schizophrenia, those cases seem of special interest that have been followed up from early childhood on into adult life. In the book, *Developmental Disorders of Mentation and Cerebral Palsies*,[11] one of us (C. E. B.) has presented 2 such cases, which reveal how some of these children who seem extremely disturbed early in childhood may come out of it in their early teens but relapse into psychotic behavior patterns after puberty. Unfortunately, space does not permit us to dwell extensively on these 2 cases.

In re-examining the three conditions, childhood autism, Heller's[6] disease, and so-called childhood schizophrenia, one finds that the patterns of Heller's[6] disease are indeed clearly different from autism and schizophrenia. To be sure, any examiner who first sees the child at around the age of 5 years without an adequate history may not be able to distinguish Heller's disease immediately, although there are some characteristic patterns. If the life history and complete observations are available, however, Heller's disease can certainly be ruled out. Less clearly defined is the difference between childhood autism and what should be called the late infantile form of childhood schizophrenia.

Those children who never overcome the severe disturbance of childhood autism are likely to end up under custodial care in institutions for retarded children, since their chronic condition does not justify admission to a mental hospital and their inability to learn places them in the group of retarded children, even if the etiology is different from the organic brain syndrome so prevalent in children with mental defect of severe degree. It seems characteristic of childhood schizophrenia that many children overcome a great many of their early difficulties under adequate care and treatment and reach much higher and often perfectly normal mental levels

in their teens. Their fate is often sealed, however, when at the time of puberty they fail to make an adequate adjustment to the increased demands of adolescence and adulthood. At that time, many of these persons show a relapse into psychotic behavior patterns. In this event, one may readily understand that the early manifestations were due to the same underlying morbid mannerisms that manifest themselves in adolescence, and these cases clearly appear to be incidences of what may be justifiably called the "morbus Bleuler."

One of the most characteristic aspects of the behavior patterns of these children is their extremely compulsive, constricted behavior. These children are likely to limit themselves to repetitious behavior patterns, which on the most primitive level are constant rocking and jumping or playing for hours with a piece of rope or some dirt they have picked up. They are not able to shift by themselves from one form of action to another and react with extreme anxiety, often violent temper tantrums, and aggressive defense.

A boy whom one of us (C.E.B.)[11] had an opportunity to observe over several years showed this kind of compulsive behavior in an exemplary fashion. He went on long walks with his male nurse or tutor, and if he left the house through the back door, he had to return the same way even after many hours; if he left through the front door, he had to return that way. He crossed the lawn in a zigzag manner, and if on the way back his companion would take his arm and draw him straight across the lawn, the child would get extremely excited, tear himself loose, run back, and follow exactly the same zigzag path he had used in the morning when leaving home. If by chance he touched some corner or furniture in the house, he had to retrace his course until he had passed the objects successfully. Dressing or undressing was a horrible experience since he repeated the same movements (such as getting his head and arms through his T shirt) for minutes and, if not interrupted, for hours, since each movement had to be done in a certain way and nothing else would satisfy him.

Observations of this boy provided much information on the compulsive behavior patterns of children of this type. They respond with anxiety and hostility if their established course of action is interrupted or changed, and yet they depend completely on someone who would liberate them from their compulsion since otherwise they get stuck in the same rut, unable to get out by their own power. This shifting of behavior has to be accomplished by the therapist and the whole environment, and is possible only through skill and understanding of the child's psychological mechanisms. Any sudden action and force are as disastrous as leaving the child to his own resources and letting him persist in his obsessive behavior.

This brings us to the question of therapy. In the treatment of childhood schizophrenia, we deal especially often with compulsive behavior patterns. To break the vicious cycle, these children must learn gradually to increase their experience. This must be managed with great care because an abrupt step will cause extreme anxiety while, on the other hand, the horizon will contract progressively if the child is left to himself, and he will gradually become one of those persons whom we were wont to see and can still see in old state institutions—sitting hunched on a bench and rocking for hours on end, or indulging in other compulsive behavior repetitions such as sucking the thumb, swaying back and forth on the floor, masturbating, or playing with some material they had picked up.

The treatment can be greatly facilitated by the use of ataraxics. We are not in a position to discuss the merits of the various ataraxics and can only say that, as

far as we know, it is still a matter of trial and error to determine the specific reaction of the individual child. A few years ago, one of us (C. E. B.) made extensive use of *Rauwolfia serpentina* (Koglucoid*), which proved very adequate with a minimum of untoward side effects.

The neuropathology of the conditions described in the foregoing is still little explored. This is due mainly to the fact that very few of the children have died, and of these, not all had adequate case histories and clinical diagnoses. In 1948, Yakovlev et al[16] gave a detailed account of the history of a child who had been thought normal up to the age of about 2 years. At that time the father enlisted in the Army and the child changed almost overnight from a happy boy to a very sad one. He deteriorated progressively, gradually losing speech and developing stereotyped motor patterns. Attractive-looking and eager, the boy gave the impression of being a normal child, although he seemed separated from his surroundings by a glass wall. At 4 years of age he was still a sturdy, well-developed, rather handsome boy. He never talked but made inarticulate sounds; in general he was overactive. The authors considered the case a characteristic instance of Heller's syndrome or dementia infantilis.

The boy died shortly before reaching his eighth birthday.† In 1952,[14] a brief report of the autopsy findings was given by one of us (C. E. B.), and attention was called to the severe cerebral degeneration and the presence of strange, large polynucleated giant cells in the cortex. It was summarized that the available evidence did not indicate any of the known forms of lipoidoses or degenerative diseases of a well-described nature, but pointed clearly to an "organic" character of the mental disorder. Whether the condition should be interpreted as "postinfectious," degenerative, or metabolic could not be stated with certainty.

Summarizing the autopsy findings and trying to avoid repetition of what was reported previously, we may say that the brain showed severe atrophy in spite of a normal weight of 1340 Gm. The leptomeninges showed extreme fibrotic proliferation, while the cortical layers were atrophic, the sulci widened, and the cortex itself sclerotic. There was a marginal subpial glial layer, and the cortical cells showed a great amount of chronic severe Nissl degeneration. As indicated by silver stains, there was an increase in astrocytic glia.

A detailed study of the brain and cerebellum revealed a number of interesting aspects not previously reported. In the cerebellum, there was a striking patchy degranulation of the granular layer, which affected all cerebellar folia (fig. 1). The Purkinje cell layer was irregular, and Purkinje cells were replaced in several places by a triangular focus of degeneration, in which a vessel was found covered with polynucleated giant cells filled with some fat and dark-staining bodies of a rather round shape, which otherwise could not be identified. In the perivascular spaces outside the cells were found some round, minute bodies similar in nature

* The trade name of Panray Corp. for powdered whole root of *Rauwolfia serpentina* containing about 20 alkaloids is Koglucoid.

† While the boy appeared fairly well until about a month before death, he developed severe stomatitis and gingivitis with swelling of the lips and ulcerations in the mucosa. During this period he ran temperatures of 100 F. and more, and lost weight rapidly.

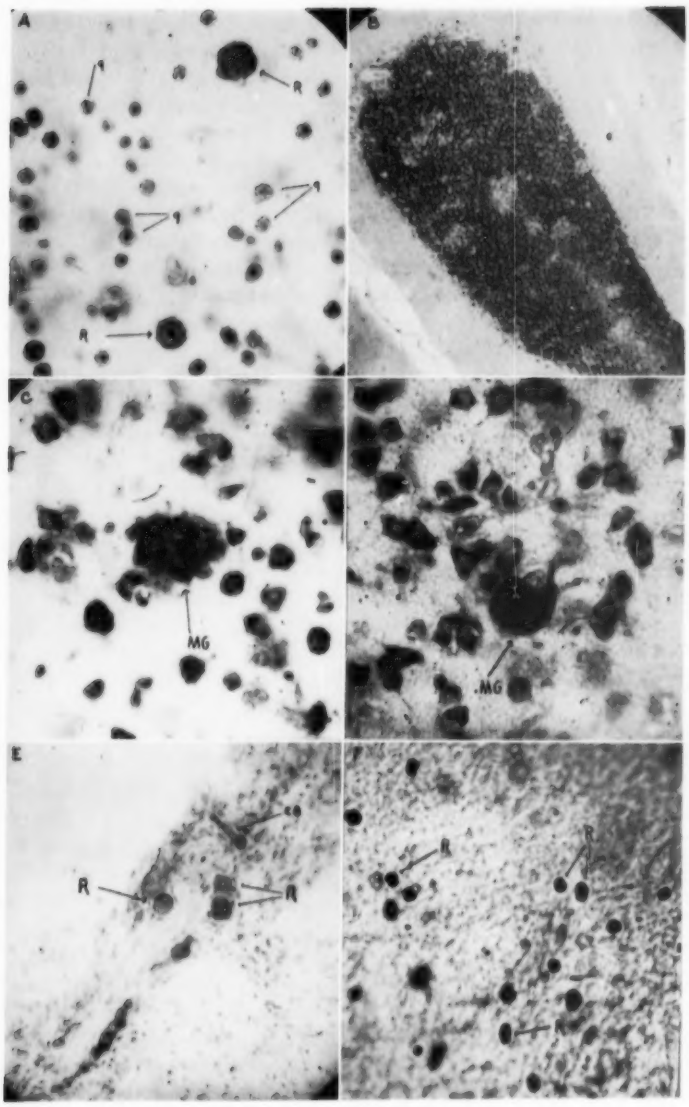

FIG. 1. *A* (hematoxylin and eosin stain), Russell bodies (R) in granular layer of cerebellum under high magnification; (g) remaining granular cells in degranulated areas. *B* (hematoxylin and eosin stain), Cerebellar leaf with marked spotty degranulation under low magnification. *C* (Nissl stain) Multinucleated giant cells (MG) with inclusion bodies of various size. *D* (Nissl stain), Multinucleated giant cells (MG) in cerebral cortex. *E*, Concentric concrements—Russell bodies (R) in subependymal tissue with calcification of capillary (ca). *F* (Schiff's periodic acid stain) Numerous concrements (R) in a degenerated brain parenchyma.

to those seen within the giant cells. Most striking in the cerebellum were several areas of granulation in which a spotty accumulation of microglia was found (fig. 2*B*). Similar areas were also found, although less frequently, in the cerebrum (fig. 3*A*). While most of these granulomas were rather homogenous, a lighter center was found in a few in which a group of smaller bodies was nested, almost like eggs in a basket (fig. 3*A*, *B*). As shown clearly in figure 3, these bodies are much smaller than the surrounding epithelioid cells, which form the body of the granuloma. In the cerebrum, some multinucleated giant cells were found in which a large number of nuclei were embedded in a large cytoplasm body. A study of the perivascular tissue revealed several areas of necrosis in which a large amount of unusually large phagocytes were found, filled with masses of inclusion bodies, which varied greatly in size and staining qualities. Comparing these phagocytes with the gitter cells so well known to neuropathology, one cannot help but observe that they are of a very different nature. While the gitter cell is usually round and filled with fat stainable with Sudan IV, the phagocytes in our case were many times larger, of a grapelike cluster, and rarely round. They were filled with large transparent bodies and smaller dark-staining materials of varying size.

Another finding worth mentioning were the numerous concretion bodies of somewhat concentric shape although irregular in surface. These bodies were similar to what has been described recently in the American literature as "Russell bodies."[17] They were not identical with the corpora amylacea of the German literature, but were pseudocalcium or neurogel[18] and did not give a positive calcium stain. In certain small areas, however, especially at the bottom of some sulci and in the cerebellum, a few vessels showed true calcification of the type recently observed by us in idiopathic familial nonarteriosclerotic calcifications, which will be discussed in another paper.

COMMENTS ON THE PATHOLOGY OF HELLER'S DISEASE

An analysis of the histopathological observations in this case confronts the investigator with almost insurmountable difficulties. The child fell ill at the age of 2 years but did not die until six years later. As a child of 6 months, he had a severe cold and high fever and was treated with sulfa drugs for several weeks. During his stay at the Walter E. Fernald State School (from 1944 to 1948), he had acute infections on numerous occasions and was treated extensively with a variety of antibiotics, especially penicillin. The child did not show any remarkable physical changes in the last four years of life except that at the time of death he was somewhat undersized (3 feet, 10 inches) although otherwise well developed. He died of chronic bronchitis, which in the final stage developed into bronchopneumonia.

Histopathological studies of the brain revealed beyond doubt that at the end of his life, the boy's central nervous system showed evidence of degeneration, cortical sclerosis, abnormal structure of the cerebellum, foci of myelitis, multinucleated giant cells, granulomas, meningeal fibrosis, and others. The evaluation of these findings is difficult, but the evidence seems to point to an infectious nature of the process, at least prior to death. The most significant findings with regard to an encephalitis were the small granulomas found in the cerebellum and cerebral white

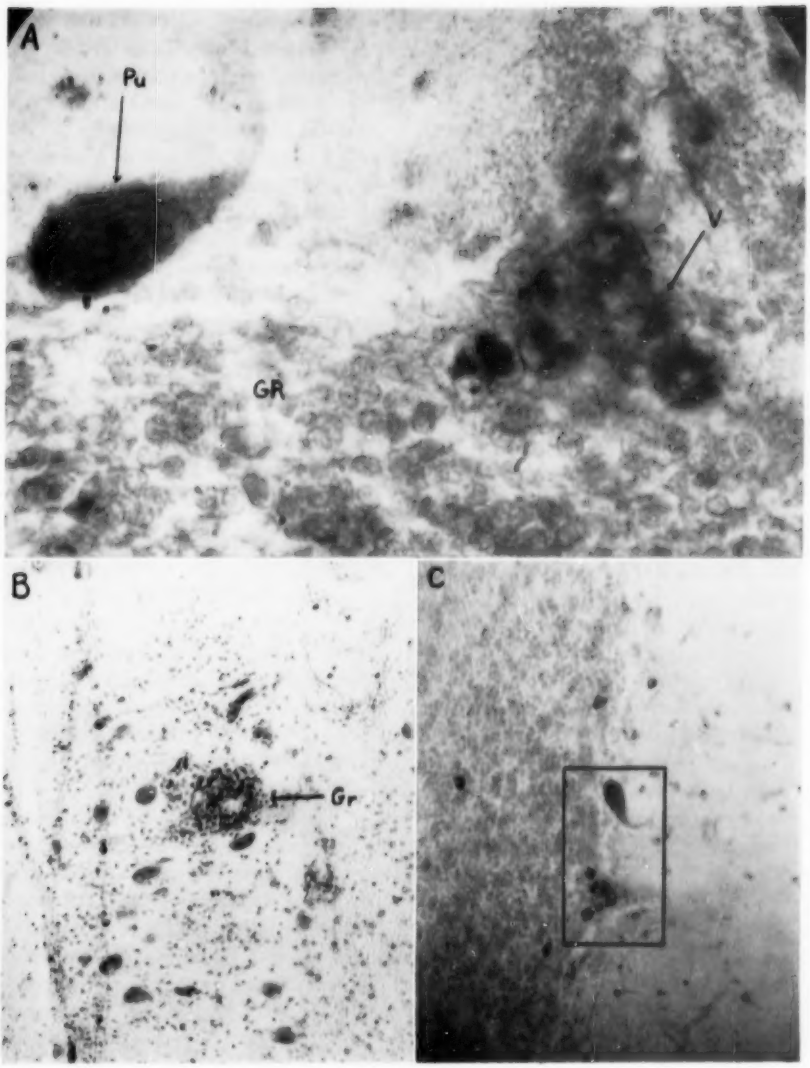

FIG. 2. *A* (Nissl stain). Purkinje cell (Pu) layer of cerebellum. Vasculitis (V) replacing Purkinje cell. Many multinucleated giant cells with inclusion bodies. (Gr = granular area.) This figure is the square area from figure 2*C* under high magnification. *B* (Nissl stain), Granuloma (Gr) in the dentate nucleus of the cerebellum. The light area in the center contains microorganisms (see figure 3). *C* (Nissl stain), Squared-off area is the same as figure 2*A* under low magnification.

matter, the vasculitis, and the presence of extremely unusual multinucleated giant cells, which were found in the immediate environment of capillaries and in the perivascular spaces of several cortical vessels. These multinucleated giant cells were not identical with the phagocytes or gitter cells found in many of our cases of encephalomalacia, cystic degeneration of the brain, or traumatic lesions.

As to the nature of the infectious process, there was no evidence of residues of a bacterial meningoencephalitis of a more common nature. It appears unlikely, therefore, that the boy experienced a bacterial infection and its sequelae in infancy or early childhood. As to a viral disease, the foci in the white matter as well as the small glial nodules and diffuse brain atrophy were compatible with such an occurrence, but it does not seem likely that a viral disease, if successfully overcome, would have left this type of neuropathology and would have produced the clinical picture of a progressive deterioration.

In recent years, more and more single reports have been published in which the effects of different fungus diseases on the central nervous system, usually combined with foci in areas like skin or lungs, have been described. Infection of the skin and lungs with *Actinomyces* has been known for many decades. Among the clinical entities now better understood are blastomycosis,[19] coccidioidomycosis,[20] histoplasmosis,[21, 22] cryptococcosis,[17, 23–25] candidiasis[26, 27] (moniliasis),[28] and most recently mucormycosis.[29] In each of these diseases, the reports have been based on clinical and bacteriological identification during the life of the patient. Neuropathological reports were based either on experimental animal infection or, in a relatively few instances, on autopsy findings. Through the previous clinical and mycological observations, it was possible to identify the foci of pathology in their specific nature. It must be emphasized, however, that the mere identification of a fungus disease from autopsy material is possible only when characteristic pathology such as meningitis, granulomas, brain abscesses, and foci of mucous degeneration is combined with the presence of a specific fungus like *Cryptococcus neoformans* (recognizable by its capsule), *Candida albicans*, or *Mucor*.

All cases reported in the literature have been relatively acute conditions arising in sick patients. The argument has not yet been settled as to whether fungus infection was the original cause of the disease or the fungus was permitted to grow under pathological conditions, often after extensive treatment with antibiotics. Since the discovery of the infantile form of toxoplasmosis, which is thought to be transmitted from mother to infant either before birth or during the process of birth, toxoplasmosis is known to be the etiological factor in some cases of infantile blindness, epileptic seizures, mental deficiency, and cerebral palsy. There are, however, many undetermined cases where diagnostic tests for toxoplasmosis are negative. Whether a fungus infection of the mother could be transmitted directly to the infant, either from the vagina or through other sources, and could produce in addition to skin lesions a subacute meningoencephalitis, which progressively leads to mental deficiency, epileptic seizures, and blindness, is still a matter of speculation. Papez and Papez[30] described mycotic organisms in a number of mentally defective patients, but otherwise no irrefutable evidence has been provided for the existence of chronic subacute meningoencephalitis of the newborn infant due to a

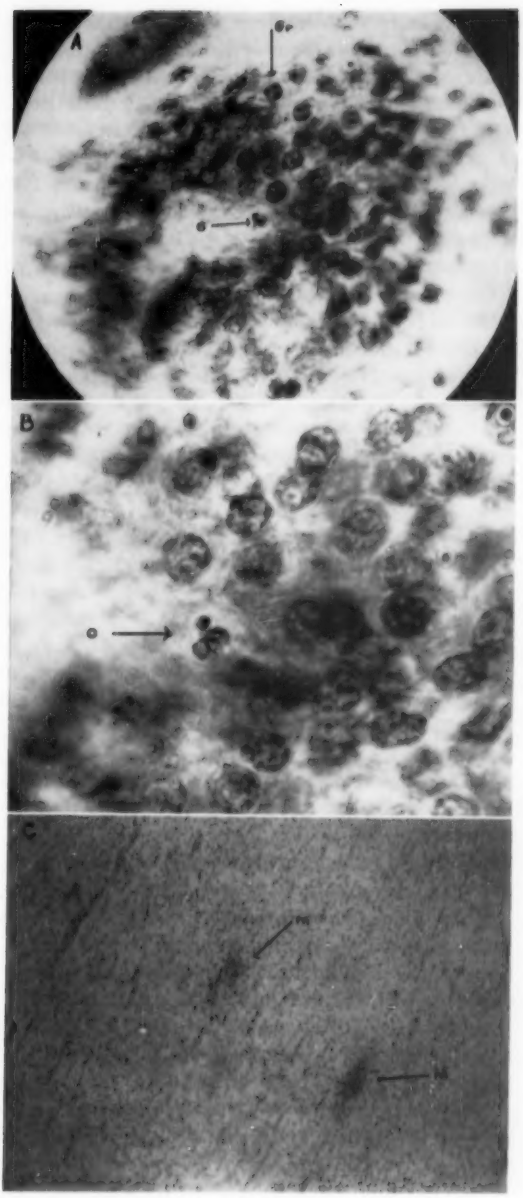

FIG. 3. *A* (Nissl stain), One of the cerebellar granulomas (Gr) under high dry magnification. Note the light center area with sporelike organisms (o). *B* (Nissl stain), Same as figure 3*A* under high magnification, oil immersion. *C* (Nissl stain), Cerebral white matter with spotty microglial reactions (m) as seen often in postvaccinal encephalitis.

fungus infection. Whenever evidence of the presence of fungus-like organisms has been found in the nervous system and there has been definite evidence of a chronic meningoencephalitis, the question arises as to whether or not the growth occurred in the terminal stage of life. If no characteristic neuropathological lesions are found, the fungus may have invaded the system after death, not to mention the possibility of a pollution of the autopsy material.

The neuropathology of our case of so-called "Heller's disease" indicated the presence of a chronic subacute infectious condition of the central nervous system. Most significant appeared to us the fibrotic proliferation of the meninges, the fibrotic organization of the perivascular spaces, the presence of microgranulomas spread through the cerebellum and parts of the brain, the presence of giant phagocytes with inclusion bodies found in the perivascular spaces and areas of softening, the vasculitis with formation of multinucleated giant cells, and the presence of small organism-like bodies found in some of the granulomas. On the other hand, a thorough investigation of the central nervous system did not provide sufficient evidence for the presence of a known metabolic disorder.

In comparing the histopathology of our case with previous reports in the literature, one may mention that Walter Freeman[31] in 1931 reported a number of cases in an article, "Torula Infection of the Central Nervous System." In this publication, Freeman described granulomas similar to that seen in our case. However, this paper—now 28 years old—encompasses a number of apparently different diseases under the term "torula." In the meantime, it has been recognized and demonstrated by the work of Littman and Zimmerman[17] that infection with *C. neoformans* or the European blastomycosis produces a rather characteristic neuropathological picture. Moreover, the organism—rather easily demonstrated by different staining methods and characterized by its large capsule—can be found within the meningitis and other areas of pathology.

None of these findings was present in our case. On the other hand, the known cases of histoplasmosis have been characterized by large multinucleated phagocytes in which the intracellular organism could be demonstrated. *Histoplasma capsulatum* is most likely to form small granulomas or glial nodules, and the organism is occasionally found in the form of round capsulated bodies or small single spores. The observations in our case are suggestive of histoplasmosis, yet not conclusive.

Although the observations definitely indicate the presence of a subacute encephalitis, it is impossible to determine the time at which a possible infection with histoplasma took place. The child frequently suffered from periods of infection in which extensive use of antibiotics was made. The child was also in a state of extreme emaciation in the last period of his life. Although the character of the pathological process cannot be demonstrated beyond doubt, the observations appear to be worth reporting—not only on account of the rarity of the disease but also in regard to future cases, where a more thorough clinical and mycological study during life may provide the correct diagnosis and means of therapy.

SUMMARY

1. Three psychiatric conditions of childhood are associated with conspicuous

alterations in mental and emotional behavior patterns without evidence of "organic brain syndrome": childhood schizophrenia, childhood autism, and Heller's disease.

2. Schizophrenia developing before adolescence is observed especially at three critical turning points of mental development and reality contact formation: the period of puberty, the period between 8 and 12 years, and early childhood between 2 and 4 years of age. While in the first two periods the diagnosis of schizophrenia can be relatively well established on account of the generally accepted symptoms (personality changes, withdrawal from outside contacts, hallucinatory experiences, extreme compulsive behavior patterns, paranoid ideas of reference, and others), the evaluation of childhood schizophrenia developing in children who have given evidence of emotional disturbance, usually from the second year of life on, is extremely difficult. The present paper deals especially with a discussion of early childhood schizophrenia.

3. Heller's disease or dementia infantilis is a rare progressive deteriorating disease, starting at around 2 years of age and leading gradually to a state of complete mental withdrawal under symptoms of mannerisms, gradual loss of speech, inability to communicate, lack of emotional expressions, but relative preservation of appearance and motor abilities.

4. The pathology of "Heller's disease" is not fully understood, and four theories are under consideration: (a) dementia infantilis as a clinical syndrome not due to one etiological factor; (b) Heller's syndrome as symptomatic of a postencephalitic condition; (c) Heller's syndrome as related to childhood schizophrenia; (d) Heller's syndrome as a metabolic disorder, possibly related to "amaurotic idiocy without amaurosis."

5. In a case presented, our autopsy observations indicated a subacute encephalitis of long duration. The pathology was characterized by leptomeningeal proliferation and fibrosis, cortical atrophy and sclerosis, chronic severe Nissl cell degeneration, patchy degranulation of the granular layers in the cerebellum, disseminated granulomas in the cerebellum and brain cortex, vasculitis, the presence of multinucleated giant cells and large grapelike phagocytic cells with numerous inclusion bodies of varying size and shape.

6. The histopathological study provided evidence of a chronic "organic" brain disease, probably originally of an infectious nature. The pathology further indicated a possibly superimposed infection with histoplasmosis.

7. Childhood autism ("the atypical child") is a syndrome of extreme aloneness and lack of communication with the environment. The occurrence of childhood autism is due to various etiological factors: (a) organic brain syndrome, possibly due to asphyxiation at birth; (b) inability of a child to form adequate reality contacts, due to parental difficulties and severe emotional deprivation in early childhood; in these children, the ego is prevented from collecting adequate reality experiences and remains in a state of separation and weak ego boundaries; we do not consider this form of childhood autism an evidence of childhood schizophrenia; (c) the early symptom of a childhood schizophrenia, which may eventually terminate in the usual patterns of adult schizophrenic behavior.

8. A differential diagnosis between childhood autism and childhood schizo-

phrenia is often difficult before 6 years of age. Schizophrenic children often become fairly well adapted during the period between 6 and 14 years of age but enter a new critical period with the approach of adolescence.

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Factors in the Management of Mental Retardation

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As many writers have observed, the mentally retarded have always been with us. Their status in our society has, however, been less stable and their treatment and management less predictable. They have from time to time been considered the "flotsam and jetsam" of society and have in some instances had the role of coddled unfortunates forced on them. Neither the status of the rejected nor that of the overprotected is helpful to the retardate or beneficial to society; both interfere with effective management and utilization of the retardate's potentials.

Since World War II the mentally retarded have received increased attention and interest. Problems related to definition, diagnosis, treatment, and training have received close scrutiny and re-evaluation. This intensification of interest is characteristic not only of those professionally or scientifically interested in mental retardation, but also of persons intimately concerned and those who are in positions of responsibility related to general social planning in areas of education, recreation, and health and welfare. The scope and significance of modern concern are well reflected in the articles of Masland[6] and Sarason and Gladwin[8] in the journal of the American Association on Mental Deficiency, May, 1958.

The physician, whose interest in mental retardation is general rather than specialized, is in a strategic although sometimes unenviable position to be of service to families and communities in their planning for the mentally retarded. The purpose of this article is to introduce and summarize what may be considered the most salient information that must be considered in making plans for the mentally retarded. In assisting the families of the mentally retarded and in consulting with welfare and other organizations, the physician will probably find this formulation of the problem and suggestions for management helpful. The selected bibliography may also be of assistance for those who desire a more intensive sample of current thinking and conclusions about the retarded.

DEFINITION OF MENTAL RETARDATION

Mental retardation generally refers to those conditions characterized by behavioral immaturity or incompetence. Generally, such conditions are described in terms of: (1) below average intelligence; (2) inadequate adaptive behavior; (3) language and thinking difficulties; (4) perceptual and motor deficiencies; and (5) other unattained biological and social skills. In order to distinguish the mentally retarded from those with other special disabilities, it must be pointed out that these characteristics occur and are not accountable for by reference to special traumatic situations, frustrations, or stress. In addition, mental retardation is present from birth or early age,

and the criteria used in making a diagnosis are in terms of deviation from the developmental and test result norms for the average child or adult in our society.

DIAGNOSIS

Although our principal concern in this paper is the management of the mentally retarded, it is obvious that management begins at recognition or diagnosis. As stated previously, the criteria for making a diagnosis are based on developmental and test result norms.

Generally speaking, a diagnosis of mental retardation will require a careful study of the whole child or adult. The following categories of inquiry or examination should be considered: (1) psychological; (2) educational; (3) social; (4) developmental history; and (5) physical history and status. Similar areas of study were suggested by Louttit[3] and Kirk and Johnson.[4] It should be noted that investigations of the individual in these physical and behavioral areas will illustrate the principle that correlation rather than compensation is the rule. This means that unless there are special deprivations or other factors operating, the individual does not usually compensate for slowness or delays in one area by significant accelerations in others but will display general accelerations, retardations, or delays. That is, within the limits of the complex and irregular rates of normal maturation, the bright individual starts out faster, proceeds more rapidly, and attains motor, social, thinking, and other skills quicker than do the average or the mentally retarded; each has his own maturational pace, and these developmental rates are good although not infallible predictors of final operating levels.

Although a final diagnosis of mental retardation can be made only on substantiating evidence from several of the aforementioned categories, it will be helpful to consider the indicators or criteria within the categories.

1. *Psychological.* Most frequently the critical information will be related to psychometric indicators of retardation. These are usually got through the use of standardized tests of intelligence. Most frequently an individual's intellectual level is expressed in terms of an intelligence quotient, which is derived from the ratio between his measured mental age and his chronological age multiplied by 100, or the quotient may be derived from standard scores. These psychometric evaluations of intelligence provide a relatively quick although limited estimate of intellectual functioning. A diagnosis of mental retardation should never be based solely on their results. Standard classifications of intelligence level in terms of intelligence quotient scores vary slightly, but those shown in table I are fairly typical.

Other tests usually administered by the clinical psychologist have been designed to investigate the individual's attitudes toward self and others, feelings, motivation, control, etc. Many of the findings from projective and psychometric personality tests are more pertinent to planning and management functions, although they may yield relevant and important information for diagnosis.

2. *Educational.* If the subject is old enough to have attended school, the educational history may be significant. Academic retardation of three years or more is usually taken to suggest probable mental retardation. On the other hand, if school

progress and standard achievement test scores indicate no educational retardation in relation to the child's chronological age, a diagnosis of mental retardation will probably be inappropriate.

Unevenness of academic performance should be carefully evaluated. Some of the more moderately retarded may perform fairly well in the mechanics of subjects requiring rote memory but do very poorly or fail in school subjects requiring abstract thinking. Severe emotional problems may produce school difficulties for some nonretarded children, but these are usually temporary and must be evaluated carefully to avoid misdiagnosis.

3. *Social.* As implied in many of the previous statements, mental retardation is a generalized condition, and the individual's social behavior and history reflect over-all slow maturation. Generally speaking, the mentally retarded person shows patterns of social adjustment and attainment characteristic of much younger persons. His interests and social skills are consistently delayed. The selection of play materials and of companions as well as interest in recreational activities may furnish important corroborative evidence of pervasive retardation. The Vineland Social Maturity Scale may be used as a guide for questioning and will give a limited picture of some of the social skills the individual usually will have acquired at various chronological ages. Lacks, delays, or problems in social interactions, self-care, and control and judgment are usually commonplace with the mentally retarded. The fact that the mentally retarded person has difficulty in going beyond details and concentrates on concrete and immediate experiences at the expense of more abstract social thinking and relationships results in the frequent picture of poor judgment and lack of appreciation of broader social implications of his own or others' actions. The mentally retarded person is also less aware of and less sensitive to the social significance of political, economic, and other events that take place around him.

4. *Developmental History.* The correlation between maturation rate of one behavioral area with others is never perfect. However, despite irregularities, which are characteristic of the retarded as well as the normal individual, the rule is still correlation rather than compensation. There will be found significant delays in the acquisition of various motor skills and other indicators of physiological maturation. Information on bowel and bladder control, self-help, talking, and dentition can usually be obtained from parents.

5. *Physical Status and Medical History.* The physical examination is necessary to

TABLE I

Typical Classification of Intelligence Level in Terms of Intelligence Quotient

Functioning level	Old designation	Intelligence quotient range
Average	—	90–110
Borderline	—	70–89
Mild	Moron	50–69
Moderate	Imbecile	25–49
Severe	Idiot	0–24

determine the individual's level of biological maturity and the existence of anatomical or physiological abnormalities or dysfunctions. Neurological, endocrinological, and other physiological complications or disturbances may be regarded as important although not exclusive determinants of mental retardation. General medical findings important in establishing etiological factors will also be relevant in planning for the mentally retarded and estimating prognoses. Physical factors may impose limitations and preclude certain arrangements, as well as point up the need for general or special medical treatment.

MANAGEMENT

As stated in its definition and basic to its diagnosis, mental retardation is characterized by developmental retardation of varying degrees. This retardation manifests itself most outstandingly in general social insufficiency so that treatment and management must be planned for with great care. The goals of such planning should be to: (1) help the mentally retarded individual make the most of his potentialities and develop to his maximum level of social competency; (2) help the individual to make his maximum contribution to society in general; this may be only at a level of simple self-help or self-care, at the level of self-maintenance under close supervision, or in terms of becoming a positive, cooperative asset in the community; (3) help the mentally retarded person secure satisfactions for his individual needs in socially acceptable ways appropriate to his level of development.

To achieve maximal results in reaching these goals for society and the mentally retarded, realistic recommendations must be made to parents and other responsible persons. In each case decisions must be made, and the following list of critical areas to be considered will serve as a guide for making decisions and planning for the effective management of the mentally retarded. These are given more or less in the order they should be considered although it must be realized that answers to subsequent questions may alter the evaluation and usefulness of previous findings. Consideration must be given to: (1) characteristics of family situations; (2) need for segregation or institutionalization; (3) capacity for self-control or requirements for social or external control; (4) community resources for special education or special training for the retarded; (5) vocational opportunities.

1. Family Situation. Careful evaluation of the adequacies of the family situation must be made. Although economic factors are not of primary importance, the burden of maintaining a mentally retarded individual should not be such as to disrupt or cripple severely the economic stability or competency of the family. Greater stress should be placed on the effective functioning of the mentally retarded in a generally effective family grouping. The sacrifice of a family to the care and protection of a mentally retarded individual is too high a price.

If the family has unrealistic expectations for the mentally retarded person, either too high or too low, and reacts with either rejection or overprotection, trouble in general adjustmental areas must be expected. Educational counseling will probably be required for parents of any retarded person if the extremes of frustration from consistent failure or overprotection from life experiences are to be avoided. The physician must be most careful to give a realistic evaluation of the mentally retarded

person and avoid the extremes of unjustified pessimism or optimism. Exaggerations in either direction are likely to result in complete rejection of the diagnosis by the parents or stimulate them to make unreasonable plans or engage in overambitious or hopeless plans for medical or educational "rehabilitation" of the retardate. The weary shopping of parents for new expert opinions is not only tragic but grossly wasteful of time and money and generally postpones the beginning of appropriate planning. The parents of the mentally retarded person must understand clearly the limits and potentialities of the retardate and the resources available in the local or general community that can be utilized for his education or training or supervision if required. They must be helped to overcome their feelings of shame and guilt and understand and accept the facts of retardation. Rheingold[7] and Kirk et al[3] presented very helpful suggestions for counseling the parents of the retarded.

2. *Segregation or Institutionalization.* A majority of mentally retarded persons spend their lives in their home communities and in the homes of their parents. However, each case must be evaluated as a possible candidate for commitment or enrollment in an institution for the mentally retarded. Institutionalization should be considered if: (1) the retardate is severely handicapped and the family is unable to care for him; (2) the local community has no special educational or training services for the retarded and the individual might benefit from such facilities within an institutional setting; such institutional placement would generally be temporary and viewed as transitional; (3) the individual retardate is unable to adjust in the community because of antisocial behavior, which might eventually prove disastrous to himself or others; this point will be considered further in the discussion of social and self-control; (4) the degree of retardation is so severe that training would not be a critical question but where adequate custodial care is the major and almost sole need; generally speaking, most communities and most homes cannot provide the specialized care and treatment that the modern institution provides through its physical and professional resources.

Institutionalization should not be recommended for the retardate whose family is able to provide for his care and supervision, and the community for his training, and who is making a satisfactory although limited and self-rewarding adjustment in his own community.

3. *Capacity for Self-Control or Need for Social Control.* As most persons develop they form notions of right and wrong, good and bad; they incorporate in their value systems the rules, regulations, and codes of society. This is a maturational process, which usually results in the internalization of controls and is generally referred to as self-control. The mentally retarded person is usually slower in attaining this type of self-control and may exhibit peculiar, inappropriate, or antisocial patterns of behavior. He is frequently more suggestible and less critical of external forces that might exert undue influence in the direction of delinquent or dangerous behavior. In no sense is he inevitably bad or antisocial. He simply requires closer supervision and a longer period of time to develop the simpler types of self-control. He frequently remains childishly immature in being unable to forego immediate satisfactions for more important and greater eventual gains and may often risk long-term goals to satisfy immediate needs. These characteristics point up the need for assisting the

retardate to satisfy his needs legitimately through appropriate means. If the retardate is unable to learn the usual techniques of self-control, provision must be made for close supervision and external controls must be provided.

4. *Community Educational Resources.* The mentally retarded person profits minimally in the ordinary academic situation. In competition with the normal or more able pupil he becomes discouraged, distressed, and frequently a burden to himself and others; his self-regard may be severely damaged, and in trying to compensate for his academic inferiority, he may develop inept and antisocial behavioral patterns.

In the past 30 years great advances have been made at a community level in the development of special educational classes and services. These services are designed to assist, among others, the mentally retarded and deficient through opportunities to develop their potentialities to a maximal level. The activities of such classes or centers do not involve coddling or overprotection of the mentally retarded but instead, setting realistic goals and providing the training required to reach them. For some retardates the goals set may seem very simple and limited; for others they may appear almost as complex and varied as those of the average person. No matter how limited or extensive the training provided, the general goals are to make the retarded person as competent as possible in areas of self-help, self-control, and social adequacy. More extensive discussion of education and training for the mentally retarded or deficient will be found in articles by Johnson[2] and Capobianco.[1]

5. *Vocational Opportunities.* The previous discussion of educational and training services implies that the mentally retarded person can be prepared for action which in some way can be beneficial to himself and society. Louttit[5] listed suitable occupations for persons of various mental ages; such a list can be very helpful in planning the training program as well as the vocational and occupational adjustment of the retarded person. The scope and range of difficulty of the jobs listed are broad, and the need for such persons in these jobs exists even in small communities. There are also public and private sheltered workshops, which provide appropriate occupations and adequate supervision for retardates of varying abilities. For some, permanent placement in such workshops is desirable; for others, the workshop may serve as a transitional or trial placement, which may prepare for eventual introduction of the retardate into the broader social and occupational world. Satisfactory occupational placement of the mentally retarded person is dependent on many factors. First, the job must match the developmental maturity and training of the retardate. It must also provide appropriate supervision and guidance, and finally, it must have significance for the individual and provide him with a feeling of importance and achievement. The job should also help him to grow in the direction of optimal self-sufficiency.

SUMMARY

The purpose of this article has been to present salient information and factors related to the management of the mentally retarded person. Because of his status and function in our society, the physician is in a particularly strategic position to utilize information on this subject. He is frequently the first professional person consulted or the second after parents or academic personnel have developed suspicions

about the development of the child. With the assistance of experts from other disciplines, he is expected to arrive at a diagnosis or definitive evaluation of the suspected mental retardate.

It has been possible to cite only briefly the important information to be considered in arriving at such a diagnosis, and these areas of evaluation are not exhaustive but only suggestive. No attempt has been made to discuss the special types of mental retardation, such as mongolism, cretinism, etc., and special etiological factors have been considered outside the scope of the present article. Management through therapeutic techniques whether medical or psychological has not been discussed here. Research into and results from such approaches have been well reviewed in the previously cited articles by Masland and Sarason and Gladwin.

The primary purpose of this article was to portray critical aspects of the problem of mental retardation management in the social context, the context with which the general practitioner will need to be familiar and utilize in his consultations regarding practical problems of such management. The behavioral equipment and repertory of the retarded person were discussed so that the retardate's problem will be neither intensified through overexpectation nor his potentialities wasted or underdeveloped through overprotection.

Since management of mental retardation begins at diagnosis and the success of management depends on the validity of the diagnosis, a final word is in order about special difficulties often encountered at this point. Especially difficult is the differential diagnosis between mental retardation and autism in children. Great care must be used in evaluating the psychological and developmental data in making a differentiation between these two states. Extreme caution in diagnosis should also be used in patients with cerebral palsy no matter what the etiology. The hyperkinetic child also poses difficult diagnostic problems. Finally, the evaluation of all borderline cases will be difficult and time-consuming.

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More Magic in Words*

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We all have seen it. With the wise luminous eyes that we have only as children, we have watched them—men, beasts, and wagons—marching into town with much noise and fanfare. With a few poles and some canvas, they promptly fence in a parcel of the earth exclusively reserved for illusion. It is a traveling circus. In the small magic space, defying the laws of physics, jugglers, acrobats, and magicians conjure a vast world of wonder. Time passes. We grow up and become perhaps physicians, perhaps writers. One day we realize that, like the performers in a circus, we too can create a world of illusion. Instead of poles and canvas, we use *words* to fence in a fragment of world, wherein illusion, like the acrobats in a circus, joyously swings through the air.

Animals use sound and motion to communicate. Only man uses sounds and signs—words—as symbols of things, events, and ideas. Words are the most precious "tools with which to make other tools." To know how to use them is man's greatest attribute. Words are the most dynamic force in medicine. Words are an instrument of healing, of creation, and of communication; of healing, because down the ages they have constituted a powerful curative agent, from the Hippocratic catharses to present-day psychoanalytical dialogue; of creation, because one single new word—such as "pneuma," "circulation," "cell," "antibiosis," "unconscious"—has often triggered a whole new system of medical philosophy; of communication, because the spoken and the written word—those two most faithful servants of thought—are the wings that permit thought to dart from mind to mind as a bird darts from branch to branch.

In our profession, obscurity in expression, because of the aura of mystery it created, was in times past the anvil on which were hammered iron dogmas that, like lead in a bird's wing, hampered the flight of medical progress. Fortunately, in modern times lucidity has become imperative in scientific language. The lucid style of Osler and Cajal only enhanced the rich content of ideas in their writings, just as the simplicity of the modern sportswoman's clothes enhances her ideal figure.

But there is another quality that is indispensable in making language not only a vehicle of thought but a captivating vehicle as well. We refer to good rhetoric, to the classical "art of good saying." The fast pace of modern life has killed the two most individual forms of word communication: the epistle and conversation, substituting instead those impersonal rockets—the telegram and the telephone call. The "art of good saying" is also too often sacrificed to the preference for stiff, prosaic, disheveled language, which clothes in drab garments or shapeless bizarre costumes

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the slender torso of scientific truth. But to degrade the language is to degrade the soul. Just as an empress is clad in a regal mantle, so must truth be clad in beauty.

It is imperative that we reinstate not only clarity but also beauty to scientific language. A little magic will bring no harm to accuracy; an image or two, a few metaphors, will like jewels add warmth and luster to cold bare facts. Thus can be mellowed the forbidding, the formidable, technical scientific language. Of course, we do not advocate a return to the old rhetoric, adipose and grotesque like the classical soprano, but to a new rhetoric, slim and athletic like a modern opera diva. This new rhetoric would help us to overcome that mental inertia which prevents us from seeing things the way they should be, that is, the way we saw them when we looked at them with a child's eyes, in all that hidden splendor which turns life into a bazaar of wonders.

This new style, already adopted by some publications, is afraid neither of literature nor of humor, and freely borrows from both to acquire charm and grace. Style *is* thought. One writes as one thinks. Many of our medical predecessors did not fear a style rich in imagery, and their prose was a magnificent Gobelin tapestry.

Let us try, then, to restore not only beauty but also joy to scientific language, using imagery and metaphor in the same manner as a fine jeweler uses tools to polish precious gems. The revival of taste for beautiful prose in science would augur for much good to the future of the *lingua medica*. Until that revival occurs, let those of us who believe in a warm, colorful language, even in science, continue our cheerful crusade. And should anyone remark that our prose wears too bright a tie, let us gently answer that it is not a tie they see but our very spine showing through.

SYMPOSIUM ON MEDICINE AND WRITING

The Symposium on Medicine and Writing that appeared in the November 1956 issue of INTERNATIONAL RECORD OF MEDICINE has been published recently as a Monograph. The articles included in this Monograph are: "The Editing of a Modern Medical Textbook" by Russell L. Cecil; "Plain Talk and Clear Writing" by Morris Fishbein; "The Principles of Bibliographic Citation" by John F. Fulton; "The Art of Communication" by Joseph Garland; "On Writing a History of Medicine" by Douglas Guthrie; and "Minerva and Aesculapius: The Physician as Writer" by Félix Martí-Ibáñez.

This 72-page Monograph is sold for $3.00. As the fourth in the series of MD International Symposia, this book is the companion piece of *Medical Writing*, which was published in May 1956.

To obtain this monograph, write to MD Publications, Inc., 30 East 60th Street, New York 22, N. Y.

BOOK REVIEWS

Family Guide to Teenage Health. EDWARD T. WILKES, M.D. New York, The Ronald Press Company, 1958. Pp. 244. Price $4.00.

Written for parents of teenagers and for teenagers themselves, this enlightened book should be most helpful in dealing with many of the common problems of adolescence.

In "Growth and Development," the author, a distinguished pediatrician who has been especially interested in adolescent problems, discusses the major factors of "normal" growth—heredity, nutrition, illness. He considers variations from the "normal" height and weight development and suggests the adequate treatments for the fat and the thin adolescent.

In "General Health" is presented an excellent review of nutrition, daily living habits, smoking and drinking, body care.

"Adolescent Ailments" considers common ailments of adolescence, ranging from acne to major diseases. Menstrual disturbances—their cause and treatment—are discussed.

The book concludes with an illuminating discussion of sex education and sex problems and of the psychology of adolescence.

This fine book is recommended for parents of teenagers, who will find, in language they can understand, a comprehensive review of the major problems of their children.

The Physician and Group Practice. Edited by EDWIN P. JORDAN, M.D. Chicago, The Year Book Publishers, Inc., 1958. Pp. 238. Price $6.75.

Group medical centers continue to increase steadily in popularity and number in the United States. This book will be welcomed by all physicians interested in learning more about group practice and also by members of groups who want to delineate some undefined areas of their relationship.

The principal advantage of group practice to the patient is that better medical care is made possible and it can be more economical, while for the physician, the advantages include opportunity for professional improvement and specialization, economic security, release from pressure of being on 24-hour call to patients, and opportunity for better service to the community. Disadvantages are also found to group practice, and not every physician would be able or would want to adapt to the relationship.

The contributors to the book, for the most part members of medical groups, have detailed economic, legal, personal aspects of group practice, covering completely every aspect that would concern the physician. Included are chapters on forms of group practice, starting or joining a group, the physician in the group, types of internal structure, methods of distributing income, business management. Two appendixes provide suggestions for data to be included in a partnership agreement and a sample form of articles of association.

This thorough book is an excellent reference for all those concerned in any way with group practice.

GOOD RESULTS REPORTED WITH ANTIMALARIAL. Prolonged treatment with the antimalarial chloroquine (Aralen, Winthrop) resulted in complete remission or major improvement in 31 of 39 cases of active rheumatoid arthritis (Brit. M. J. 2:1199, 1958). In 12 patients a full remission was obtained, with weight gain and return of erythrocyte sedimentation rate to normal. A major response, grade 2, was observed in 19 patients. All patients were treated for periods ranging from three months to four years.

NEW ASPIRATOR. A new aspirator that is portable, requires no electricity, and maintains a high vacuum was recently announced by Conitech, Ltd., Chauncey, N. Y. Featuring a unique foot pedal action, this Farr Aspirator can be set up in less than a minute. It weighs only 10 pounds, yet provides sufficient suction to clean out thick material deposits.

INFANT SURGERY. The best time to perform surgery on the newborn infant is during the first 72 hours of life, said Dr. H. William Clatworthy, Jr. (Columbus, Ohio), at the A.M.A. clinical meeting in Minneapolis. The patient is in an excellent nutritional state during the first 72 hours with high levels of hormones and inherited antibodies to enable him to adapt to stress and thwart infection. He also requires less anesthesia because of his high pain threshold.

CONTRAST MEDIUM. Administered intravenously to permit simultaneous visualization of the renal and biliary tracts is methylglucamine diatrizoate iodipamide (Duografin, Squibb). The drug provides complete visualization of the entire biliary tract, even in cholecystectomized patients, and of the kidneys and urinary passages, including the renal pelvis, ureters, and bladder.

RADIATION THERAPY. No significant genetic damage attributable to radiation has been found in 644 married women treated with roentgen-ray therapy to correct sterility, it was reported by Dr. Ira I. Kaplan (New York City) at the meeting of the Radiological Society of North America in Chicago recently. No incidence of sterility to successive generations of the offspring of the study group treated by roentgen-ray therapy of the ovaries and pituitary has been noted. To date 14 men and 20 women, children of the original patients, are married and 31 of these have borne 45 normal children. The original patients were followed from 1 to 33 years.

ANTIALLERGY DRUG. Of 100 patients treated with a new antihistaminic compound, parabromdylamine (Dimetane, Robins), a halogenated monoamine, 88 per cent had a satisfactory response; best results were obtained in hay fever, allergic rhinitis, urticaria, and eczema; bronchial asthma did not respond so well (AM&CT, Jan., 1959). The drug was well tolerated except for occasional mild drowsiness on higher dosages.

BACTERIAL TEST. A fast, economical test to determine which antibacterial agent inhibits the growth of a patient's disease organisms is now available (Medikit by Media). The physician streaks a plate with a swab of a fresh specimen, places a special antibacterial ring on the plate and incubates the culture for 12 to 24 hours (up to 72 hours for certain nose and throat cultures). Observation under a light will tell him which agent inhibits the growth of the infectious organism.

BILATERAL VASOVASOTOMY. A series of nine bilateral vasovasotomies were performed on infertile patients in whom the vas deferens had been surgically severed, reported Dr. Samuel I. Roland (Palo Alto, Calif.) at the Pacific Coast Fertility Society meeting. This was the first report of the procedure without the use of an indwelling intraluminal splint postoperatively, he said. Five of the patients had postoperative semen counts of more than 20,000,000/cc., 1 had a consistently low count less than 1,000,000/cc., and 3 remained azoospermic.

CORYZA PALLIATIVE. A new antibiotic-analgesic-antihistaminic compound contains triacetyloleandomycin, phenacetin, caffeine, salicylamide, and buclizine hydrochloride (TAO-AC Capsules, Roerig). Intended for symptomatic relief of the common cold and prophylaxis against secondary complications due to commonly associated pathogenic bacteria, the drug is reported to cause minimal interference with the gastrointestinal flora.

BATS RESPONSIBLE IN HISTOPLASMOSIS EPIDEMIC. The responsible organism in a family epidemic of histoplasmosis in Maryland was isolated from 50 of 105 soil samples collected on the premises, particularly in soil adjacent to the building's foundation (Pub. Health Rep. 73:590, 1958). The house sheltered a colony of brown "house bats," and bat dung was found to be the contaminating material. This and other evidence from the literature suggest that bats may be the ecological factor responsible for a widespread histoplasmin sensitivity and many instances of urban histoplasmosis.

QUARTERLY REVIEW OF OPHTHALMOLOGY

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FOREWORD: A New Policy

The objective of the Quarterly Review of Ophthalmology is to bring together in one publication a concise record of current happenings, both national and international, in this field. Selection and editing of material are designed to prove informative to *the entire medical profession* as well as to ophthalmologists. Manuscripts for the Quarterly Review of Ophthalmology should be submitted to Dr. Arnold S. Breakey, Ophthalmological Foundation, 111 East 59th St., New York, N. Y.

HISTORICAL VIGNETTES. I.

Three Greeks: Their Impact on Ophthalmology

HIPPOCRATES, ARISTOTLE, GALEN

George E. Arrington, Jr., M.D.

RICHMOND, VA.

The roots of Western civilization are found in ancient Greece. Similarly rational thought, which culminated in modern science and medicine, springs from that source. Ophthalmology, as yet unnamed and undisciplined, had its most decisive formulation in the minds of three Greeks: Hippocrates, Aristotle, and Galen.

It must be understood that the approach to the problems of man and his nature during the age of Greece was quite different from our own. Their strength was in clear, logical analysis and abstract philosophical evaluation while concrete applied therapeutics was still in its infancy. However, consider their achievements as exemplified in these three figures.

Hippocrates (460?-377 B.C.) was born on the island of Cos, son of Heraclides, an Asclepiad who claimed descent from Aesculapius, the Greek god of medicine. Hippocrates broke the tradition of the magic medical priesthood and turned to man himself as the intrinsic site of disease. His physiology was humoral and his therapy natural, depending on nature to heal itself (*vis medicatrix naturae*). His greatest contribution and his claim as the father of modern medicine, including ophthalmology, lie in his grasp of the significance of the historical concept of disease[1]—disease as a chapter of the patient's life history. The Hippocratic school recognized and treated ocular inflammations such as trachoma (*aspritudo*), which they treated with abrasion (*ophthalmoxipis*), cauterization, and peroxide of copper. Other ocular diseases considered were pterygium, chalazion, trichiasis, entropion, amaurosis post hemorrhagicum, nystagmus, and crossed eyes. There was a confusion of glaucoma and cataract, the term "*glaucosis*" referring to cataract, which in humoral pathology was considered to be an inspissated humor in a presumed empty space (*locus vacuus*) believed to be present between the lens and iris. This error persisted for hundreds of years. But more significant was the persistence of Hippocratic emphasis on the patient himself and the code of ethics attendant on this new physician-patient relationship.

Aristotle (384-322 B.C.), also the son of an Asclepiad, of Stagira, was prominent in ophthalmological history as a naturalist with philosophical predilection. As the pupil of and next great philosopher after Plato, he also considered the problems of vision and became the opponent of his teacher's views. Plato had evolved the emanation theory of vision ("fire" flows out of the eyes bending with external "fire"), thus placing man at the center of visual function. In this, Plato embodied his view of visual function within his general dictum of "like to like" and the

EDITOR'S NOTE: This is the first of a series of articles on the contributions to ophthalmology of great men in history. Dr. Arrington is eminently qualified for the task for he has made a prolonged study of the subject and has written *A History of Ophthalmology* (New York, MD Publications, Inc., 1959).

supreme position of the soul in man. For Aristotle, since one cannot see in the dark, vision was the result of inflowing influences, the eye deserving its role as the organ of vision only insofar as it is an "actualized," functioning, integral part of man.[2] Aristotle contributed much knowledge to ocular anatomy and comparative ocular anatomy, and he recognized far- and nearsightedness and the weak sight of old age, which is evidence of his organized search for facts. But his greatest contribution to ophthalmology was to shift interest to the eye as an organ receptive of external influences. He said, "What perceives is of course a spatial magnitude, but we must not admit that either the having the power to perceive or the sense itself is a magnitude; what they are is a certain ratio or power in a magnitude."[3] Related to this approach, the laws of optics evolved later.

Galen (fl. A.D. second century) was born the son of a wealthy architect, Nicon, at Pergamum. He studied medicine in Greece and Alexandria. He traveled extensively and came to Rome in A.D. 164, soon becoming physician to the emperor, Marcus Aurelius, and many other prominent Romans. Galen is known as the most eminent physician, author, and practitioner of his era. Although he was principally an Aristotelian, he knew and criticized philosophical disciplines of Plato, the Stoics, and the Epicureans while he held Hippocrates in high esteem. However, his chief teacher was nature itself, and his anatomical and physiological views, including those of ophthalmological importance, were incorporated in his strong teleology, which rendered them acceptable to the church. His teachings subsequently became medical dogma for 1500 years. Galen considered the crystalline body to be the essential organ of vision with the retina as a netlike extension of the brain to perceive the alterations in the lens and to convey these impressions to the brain. Vision resulted from a visual spirit (*pneuma psychicon*) produced by the brain and conveyed by the optic nerves to the eyes where it "radiated outward" (Platonic), but he spoke elsewhere of the "*incidence of the radiant brilliancy*" (Aristotelian). Galen contributed to physiological optics the idea of binocular visual fields as two converging cones, and he related this concept to the anatomical form of the optic nerves and chiasm. His therapeutics embraced the Hippocratic idea of treating disorders by their opposites (*contraria contrariis curantur*). Galen introduced many ocular therapeutic measures for the removal of conditions foreign to the healthy eye. His therapeutics were based on humoral physiology, and he evolved the most advanced anatomy until his day.

To the Greeks, then, ophthalmology owes the structure of medical thought with emphasis on disease as a chapter in the life history of the patient (Hippocrates), the first naturalistic approach to the eye and vision based on observation of human experience and investigation of man both philosophically and scientifically (Aristotle), and the first practical and successful application of these principles to ocular anatomy, physiology, and clinical medicine (Galen).

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Questions and Answers

Q. *Why is my eye bloodshot?*

A. Redness of the eye may be due to subconjunctival hemorrhage, ecchymosis, or petechiae, or to passive or active hyperemia. It may be caused by superficial or deep congestion or both. It may indicate pathology of the conjunctiva, cornea, sclera, uvea, or the drainage system of the eye or their combination. Its etiology may be local or systemic.

It is fortunate for the examining physician that redness of the eye is rarely the only sign of an eye condition. Besides the history, systematic examination of the appendages of the eye and all accessible eye structures (even without extensive ophthalmological equipment) will usually clarify the differential diagnosis of the cause of ocular redness. In doubtful cases, of course, only combined clinical and laboratory studies will ensure an accurate diagnosis and proper management of the condition.

Acute conjunctivitis is undoubtedly the most frequent cause of an inflamed eye. Burning, tearing, irritation, and/or foreign body sensation is the presenting symptom. Vision is normal (or unchanged). Conjunctival congestion is more marked on the lids and in the fornices than in the bulbar conjunctiva. It is superficial in type, i.e., it is bright red in color and it can be milked away with pressure or blanched with solution of adrenaline (1:1000); blood vessels are movable with an applicator and irregularly distributed. Discharge may be purulent (yellow) or mucopurulent (whitish) in appearance. It is more abundant in acute bacterial infections than in viral, in which case it is usually scanty. It is stringy in allergic or diplobacillary conjunctivitis. In more virulent bacterial involvement, pseudomembranes or even membranes may appear, while subconjunctival ecchymoses may be associated with conjunctivitis due to influenza, pneumococcus, or Koch-Weeks microorganisms. The cornea is usually clear (except if secondarily involved in later stages of conjunctivitis), and pupil and iris look normal. The preauricular glands may be enlarged or, rarely, tender (especially in viral forms), while the eye is never tender to touch. Occasionally, pressure on the lachrymal sac may cause regurgitation of purulent material, thus pointing to lachrymal origin of conjunctivitis.

In allergic reactions the conjunctiva is usually pale, but it may be red in dermatoconjunctivitis, a form associated with eczematoid dermatitis of the skin of the lids caused by drugs, industrial chemicals, cosmetics, or wearing apparel.

In every case of conjunctival congestion, the possibility of the presence of a foreign body must be considered. Pain in the eye in such a case is localized by the patient in one spot (actually due to corneal irritation), which frequently helps to differentiate this type of conjunctival irritation from other forms of conjunctivitis, in which patients complain of the sensation of a migrating foreign body.

Subconjunctival hemorrhage, both post-traumatic and spontaneous, can be easily differentiated from ecchymoses complicating severe types of conjunctivitis. In cases

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Note: Questions from our readers are invited. Address George Z. Carter, M.D., Ophthalmological Foundation, 111 East 59th St., New York, N. Y.

of traumatic subconjunctival hemorrhage, the possibility of a scleral perforation or rupture, covered by the hemorrhage, should be kept in mind.

Specific treatment of conjunctivitis can be administered only with the aid of laboratory procedures, including examination of conjunctival smears and cultures (for determination of the microorganisms present and the cytology of the exudate) and general systemic evaluation. Local treatment is adequate for most forms of infectious conjunctivitis. Here preference should be given antibiotics that are considered too toxic for systemic treatment, so that sensitization of the patient to an antibiotic that may later be needed for systemic treatment can be avoided. Bacitracin, neomycin, and polymyxin are such antibiotics. Sulfonamides in the form of solution or ointment have been widely used in the treatment of infectious conjunctivitis. These are certainly most useful drugs in a great majority of cases. Recently, however, attention has been called to their antigenic properties resulting in local sensitivity reactions of both conjunctiva and the skin of the lids.

Lack of a definite response to the recommended treatment within two to three days indicates that the wrong medication was used or that the offending organisms have developed resistance to the administered drug. If the infection becomes worse, the possibility of local hypersensitivity must be considered.

In the treatment of allergic conjunctivitis, the use of cortisone and its congeners has superseded most of the formerly used agents. The dramatic improvement after steroid therapy has led, however, to its rather indiscriminate use. It should be stressed that topical administration of steroids is considered by many ophthalmologists a possible cause of increased frequency of dendritic keratitis and fungus infections of the eyes in recent years.

George Z. Carter, M.D.
New York, N. Y.

Reduction in New Cases of Blindness among Children

Remarkable progress has been made in reducing the number of new cases of blindness among children, according to the statisticians of the Metropolitan Life Insurance Company. Not only has blindness due to infectious diseases decreased sharply among school-age children, but loss of sight due to the administering of oxygen in high concentration to premature infants has become rare, the report continues.

Persons in the United States who are either totally blind or with impairment of vision sufficiently great to prevent normal activities number 345,000, which is at the rate of 2/1000 of the population. New cases are estimated at 30,000 yearly, or 17/100,000 population, the report continues. Slightly more than half of these blind are 65 years of age or older, approximately 30 per cent are between ages 40 and 64, 12 per cent between 20 and 39, and 7 per cent less than 20.

ABSTRACTS

OPHTHALMOLOGY PER SE

1. *Epithelial Invasion of the Anterior Chamber.* ELLEN F. REGAN. Tr. Am. Ophth. Soc. 55:741, 1957.

Three types of epithelial growth in the anterior chamber are recognized: (1) pearl tumors of the iris, (2) post-traumatic cysts of the iris, and (3) epithelization of the anterior chamber. Epithelium may invade the chamber by implantation of free or attached cells or by spreading along an irregular incision. Epithelium will not grow by implantation in the aqueous but must come into contact with the iris. Epithelial invasion must be avoided by proper closure of the incision and is a frequent sequel to cataract surgery, a rate as high as 29 per cent being reported by one surgeon.—*Eugene M. Blake, M.D.*

2. *Gonioscopy of Recently Healed Cataract Incisions.* H. E. THORPE. Tr. Am. Ophth. Soc. 55:773, 1957.

This study deals with gonioscopic observation of 318 consecutive cataract extractions done by keratome and scissors and with multiple preplaced appositional sutures. Examination by slit lamp was made before and after surgery. Gonioscopy showed that the anterior chamber was widened by an average of 1.6 mm. Vitreous herniation after the intracapsular extraction was seen in 88 per cent and vitreous detachment in 80 per cent. Gaping of the incision was not present in 76 per cent of cases at the end of 10 weeks. Combined extraction showed a greater incidence of peripheral synechiae than in cases with peripheral iridectomy.—*Eugene M. Blake, M.D.*

3. *Congenital Ectropion and Distichasis.* GUILLERMO PICO. Tr. Am. Ophth. Soc. 55:663, 1957.

The literature contains only 12 recorded cases of congenital ectropion and there are not many more of distichia. The two may be combined and 1 such case is recorded, together with the history of three generations. Histological examination of specimens of 2 cases showed the absence of meibomian glands and the entire absence of the tarsal plates or presence of vestigial structure.—*Eugene M. Blake, M.D.*

4. *Technique in Retinal Detachment Surgery.* J. B. HITZ. Tr. Am. Ophth. Soc. 55: 477, 1957.

The author gives an excellent review of the various techniques that have been employed in operations for separation of the retina. The numerous procedures indicate that no single technique fulfills all the necessary requirements. The essentials are the induction of an adhesive or exudative choroiditis properly placed and with the retinal opening corresponding with the scleral site.—*Eugene M. Blake, M.D.*

5. *Tumors of the Orbit.* CHARLES E. ILIFF. Tr. Am. Ophth. Soc. 55:505, 1957.

This important field of diagnosis and treatment is extensively reviewed and covers the previous literature. The author's personal experience is presented, and emphasis is placed on whether the tumor arises in the orbit or is an extension of an intracranial lesion. Improved diagnostic methods presented have been successfully used in making a differential diagnosis.—*Eugene M. Blake, M.D.*

6. *Idiopathic Atrophy of the Epithelial Layers of the Iris and Ciliary Body.* H. G. SCHEIE AND H. W. FLEISCHHAUER. Tr. Am. Ophth. Soc. 55:369, 1957.

This paper reports on 97 eyes with dissemination and depositions of the pigment of the anterior segment of the eye. The pigment appears to arise from the epithelial layers of the iris and the ciliary processes from which it is freed by idiopathic atrophy of these structures. Transillumination of the eye shows iris atrophy in patches. Glaucoma occurred in 42 eyes, presumably through obstruction to the outflow of aqueous by pigment deposited in the angle.—*Eugene M. Blake, M.D.*

7. *Isolated Inferior Oblique Paralyses.* HAROLD W. BROWN. Tr. Am. Ophth. Soc. 55: 415, 1957.

The article is an analysis of 97 cases of isolated paralysis of the inferior oblique muscle, of which 6 were of unknown etiology, 70 were congenital, and the others of unknown cause. The explanation of the syndrome in most cases is a congenital paralysis of the inferior oblique muscle caused by a short anterior tendon sheath of the superior oblique muscle. Suggestive treatment is presented.—*Eugene M. Blake, M.D.*

8. *Adhesion of the Lens Capsule to the Hyaloid Membrane and Its Relationship to the Intracapsular Cataract Extraction.* A. B. REESE AND J. A. C. WADSWORTH. Tr. Am. Ophth. Soc. 55:239, 1957.

The authors have carefully reviewed the literature on the debatable question of the adhesion of the posterior capsule of the lens to the front of the vitreous body. Wide experience with the intracapsular extraction of cataract and a profound knowledge of the histology of the parts warrant the conclusion that adhesions between the posterior capsule of the lens and the anterior hyaloid occur in sufficient degree and frequency to be reckoned with in performing an extracapsular cataract extraction.—*Eugene M. Blake, M.D.*

9. *Postoperative Care of Retinal Detachment.* J. W. JERVEY, JR. Tr. Am. Ophth. Soc. 55:317, 1957.

The author feels that prolonged occlusion of the eyes, the use of pinhole glasses, and marked restriction of bodily movements not only are not conducive to reattachment of the retina but may actually have a deleterious effect on healing. When healing occurs satisfactorily under the method of greater freedom, one may feel opti-

mistic over the regime, but when reattachment fails to occur, it would seem that one would question the practice. However, Dr. Jervey's results were very good, in fact as good as those with patients treated in the more conservative manner.—*Eugene M. Blake, M.D.*

10. *A Clinical and Pathologic Study of Exfoliation of the Lens Capsule.* HAROLD GIFFORD, JR. Tr. Am. Ophth. Soc. 55:189, 1957.

The material presented is a clinical study of 62 patients with exfoliation of the lens capsule. Information was obtained by following cases for several years and from photographs of early changes. Some new pathological evidence is based on a different technique for staining the lens capsule. The author suggests a frequent dilatation of the pupil to aid better understanding and for therapeutic effect, since this act may sweep away the exfoliated material and prevent clogging of the trabeculae. The article is profusely illustrated.—*Eugene M. Blake, M.D.*

11. *Operating Room Gonioscopy in Angle Closure Glaucoma Surgery.* ROBERT N. SHAFFER. Tr. Am. Ophth. Soc. 55:59, 1957.

If permanent peripheral synechiae are present, an iridectomy may not be sufficient to obtain cure of the hypertension. Ordinarily the choice of surgery has been based empirically on the history of previous attacks or on the present acute attack. The author has found that the use of the gonioscope on the operating table, after performing an ab externo iridectomy makes it possible to determine what procedure to undertake.—*Eugene M. Blake, M.D.*

12. *Delayed Recurrences of Malignant Melanomas of the Bulbar Conjunctiva.* PHILIP MERIWEATHER LEWIS AND LORENZ E. ZIMMERMAN. Tr. Am. Ophth. Soc. 55: 109, 1957.

Since malignant melanomas of the conjunctiva are very rare ocular tumors, there is great difficulty in deciding the proper treatment in a given case. This has varied from simple conjunctival excision to enucleation of the eyeball and exenteration of the orbit. The authors report 7 cases and discuss the clinicopathological stages, with long periods of follow-up. They emphasize that, at present, there is no such study on record, hence not sufficient factual data on which to base conclusions regarding surgical management. The outcome of these cases was as follows: (1) malignant melanoma of the socket, (2) recurrence after two years but none further for 23 years, (3) metastasis after eight years, (4) recurrence after eight years, (5) recurrence after four years, (6) enucleation after eight years, (7) simple excision and lived for 15 years.—*Eugene M. Blake, M.D.*

13. *The Thermal Effect on Ocular Tissues of Surgical Diathermy Currents Used in Treatment of Detached Retina.* S. RODMAN IRVINE AND HENRY A. KNOLL. Tr. Am. Ophth. Soc. 55:123, 1957.

The authors conducted research to determine the basic electrical phenomena of

electrocautery of the eye. The results of variation of the output characteristics of the Walker Unit are documented by graphs and charts.

To avoid damage by diathermy, the surgeon must know his own machine and must have clinical experience judging the reaction of the sclera and retina. It is recommended that one should start treatment with production of too little effect, increasing to the desired effect. Such caution must be observed even when conditions appear to be more controlled because of lamellar resection and treatment of the choroid under direct observation. Any treatment must be constantly controlled by observation of the retina.

The ideal apparatus for surgery of detached retina should be constructed to deliver galvanic current and both sustained and dampened-wave high frequency current. This would facilitate production of chemical, fulgurating, coagulating, and cutting effects, all of which have a place in the surgery of detached retina.—*Eugene M. Blake, M.D.*

14. *The Human Electroretinogram.* LORRIN A. RIGGS. A.M.A. Arch. Ophth. *60*:739–753, Oct., 1958.

After a brief historical note on the development of recording techniques, the author reviews the recent data on the components of the electroretinogram (ERG). There are scotopic and photopic components of the human ERG depending on the state of light adaptation of the subject. With dark adaptation and using a large stimulus light, the recorded ERG appears to be a function of the rod system of the retina. In records made under these conditions, no information is available on the function of the cone system of the retina. Such records reflect only the state of the scotopic system of the retina.

In the last 15 years, the photopic components of the ERG have been recorded by use of several techniques such as use of red light, light-adapting the subject, flashing light of short duration, use of flicker at relatively high frequencies. Although this new approach is of clinical value, the author cautions that "... the ERG is a mass response and cannot be used for any such purpose as perimetry or measurement of visual acuity."—*Carl Kupfer, M.D.*

15. *Problems in the Diagnosis and Treatment of Lens-Induced Uveitis and Glaucoma.* P. A. CHANDLER. A.M.A. Arch. Ophth. *60*:828–841, Nov., 1958.

The literature dealing with lens-induced uveitis and glaucoma is reviewed and 12 case reports are presented to illustrate these complications. Free lens substance in the eye may give rise to little or no inflammatory reaction. On the other hand, it may give rise to a severe prolonged reaction. Such cases include hypermature cataracts, whether in situ or dislocated into the vitreous; advanced immature cataracts; traumatic cataracts; and after extracapsular cataract extraction. In these conditions, the clinical diagnosis is made by the presence of a severe prolonged uveitis and glaucoma that rarely respond completely to conservative therapy. In only 1 case was conservative treatment completely successful. The timely removal of the lens or lens matter will arrest the process, as well illustrated in the reported

series of cases. A delay in removal of the lens often renders the operation more difficult. If the lens substance is not removed, the eye will usually be lost or severely damaged.—*Ralph Z. Levene, M.D.*

16. *Further Studies on Facility of Flow Through the Trabecular Meshwork.* W. M. GRANT. A.M.A. Arch. Ophth. *60*:523–533, Oct., 1958.

The influence of opening or removing the trabecular meshwork on the facility of outflow of intraocular fluid was investigated in 16 enucleated normal human eyes by an improved technique. It was calculated that the trabecular meshwork as a whole in its relaxed state in these eyes was responsible for approximately 75 per cent of the total resistance to outflow of fluid from the anterior chamber. These results differ from the tentative conclusions originally presented by the author after earlier investigations with a cruder technique. In the present method, a central corneal hole was used to introduce instruments for dissection of the trabecular meshwork under direct microscopic view. The hole was then tightly sealed for perfusion studies of the angle. The methods of trabeculotomy or trabeculectomy included teasing with the tip of one blade of a toothless forceps or cutting with a von Graefe cystotome or a Barkan goniotomy Knife. In every eye of this series, a significant increase in the facility of outflow followed dissection of the meshwork. In normal eyes, opening the meshwork in only one sixth of the circumfereace provided an increase of approximately 50 per cent in facility of outflow. However, in trabeculotomy, a tendency for a cut in the meshwork to be self-sealing was observed, and this is one of the factors to be considered and controlled in applying the results of the study to the surgical treatment of glaucoma.—*Ralph Z. Levene, M.D.*

17. *The Adhesion of the Lens Capsule to the Hyaloid Membrane.* A. B. REESE AND J. A. C. WADSWORTH. Am. J. Ophth. *46*:495–498, Oct., 1958.

The authors' attention was directed to adhesions between the lens capsule and the anterior hyaloid membrane of the vitreous at the time of operation on supposedly uncomplicated cataracts. In order to study these adhesions, extractions were performed on 20 fresh eyes obtained at autopsy or enucleated for choroidal melanomas. In 25 per cent of the eyes the hyaloid was so firmly adherent that it came forward with the lens after the zonular membrane was broken. The adhesions may be fostered by a previous inflammation or more often may be on a developmental basis. Failure to appreciate these adhesions at the time of lens extraction is one important cause of capsule rupture or vitreous loss. When this syndrome is appreciated, it is sometimes possible to tease the two structures apart by wedging a spoon between the hyaloid and capsule with the cornea intervening. Alternatively, the use of an erysiphake allows gradual rotation and eversion of the lens and gradual separation of the two structures.—*Ralph Z. Levene, M.D.*

18. *Application of Poiseuille's Law to Aqueous Outflow.* W. K. MC EWEN. A.M.A. Arch. Ophth. *60*:290–294, Aug., 1958.

By using data derived from anatomical and physiological studies on the circulation

of the aqueous humor, the author has been able to apply Poiseuille's law to the outflow of aqueous humor. Several interesting conclusions are drawn. It appears probable that the major resistance to the outflow is not in the trabeculae or collector channels but in a low porosity tissue barrier located very near the anterior chamber side of Schlemm's canal. This barrier averages one hole (2 micra in diameter) every 0.01 sq. mm. A very small decrease in the diameter of these holes, too small to be detected histologically, could account for open-angle glaucoma.

Such an approach to the problem of aqueous humor circulation is worth while considering and should be pursued in order to quantify further the factors involved in the rate of outflow of aqueous humor.—*Carl Kupfer, M.D.*

19. *The Decline in Aqueous Secretion and Outflow Facility with Age.* B. BECKER. Am. J. Ophth. *46*:731–736, Nov., 1958.

In an analysis of 909 normal eyes, tonography revealed a significant progressive decrease in average outflow facility with age from an average of 0.33 for eyes of persons 40 years of age or less to 0.23 for those of persons older than 60 years. This was associated with a significant sharp decline in average rate of aqueous secretion after the age of 60 years. Intraocular pressure was therefore maintained reasonably constant at all ages. In vitro perfusion measurements in 54 normal eyes as well as fluorescein turnover studies confirmed the increasing resistance to outflow with age. These findings correlate well with the histological picture of ciliary body and outflow drainage channels. In 910 eyes with chronic simple glaucoma, a significant decrease in secretory rate with advancing age was also found. On the one hand, this hyposecretion may prevent pressure damage in spite of the outflow disorder. On the other hand, the same decline in secretion with age makes detection of glaucoma so difficult when single pressure measurements alone are used. With regard to the mechanism of the decrease in facility and secretion rate, it is not possible to establish cause and effect. Intraocular pressures greater than 20 mm. of mercury occurred in only 7 per cent of normal eyes, and therefore a scale reading of 4.0 with the 5.5 Gm. weight suggests a careful evaluation for glaucoma.—*Ralph Z. Levene, M.D.*

NEURO-OPHTHALMOLOGY

20. *Ocular Signs of Meningioma.* F. E. NEWELL AND T. C. BEAMAN. Tr. Am. Ophth. Soc. *55*:297, 1957.

The meningioma is the blind connective-tissue neoplasm that may arise from the meninges of any part of the central nervous system. Histological studies were made on 143 patients and the symptoms peculiar to the particular region affected are clearly reported with special emphasis laid on the ocular signs and symptoms. Diagnosis is greatly aided by roentgenographic examinations. Meningioma should be considered a likely cause of blindness in middle-aged persons with progressive atrophy of the optic nerve not arising from glaucoma, vascular disease, or syphilis. —*Eugene M. Blake, M.D.*

21. *The Concept of a Semidecussation of the Optic Nerves.* C. WILBUR RUCKER. Tr. Am. Ophth. Soc. 55:87, 1957.

This is an historically interesting article and well worth reading. One can do no better than to quote the author's summary, as follows:

> The concept of a semidecussation of the optic nerves originated with Isaac Newton in 1704 and furnished him with the explanation of single vision with the two eyes. Vater and Heinicke, 1723, two decades later and without knowledge of Newton's hypothesis hit upon the same idea and used it as an explanation of the phenomenon of half vision. Without knowledge of either of these reports Wollaston (1824) a century later, on the basis of his transient loss of vision to one side, also conceived the idea of a semidecussation at the chiasm. His publication aroused wide-spread interest in the concept and led to attaining practical significance.

—*Eugene M. Blake, M.D.*

22. *Some Neuro-ophthalmological Problems.* ALAN MOONEY. Brit. J. Ophth. 42:129–156, March, 1958.

The author emphasizes and elaborates on the useful contribution that the ophthalmologist may make to the neurosurgeon and presents a number of observations and conclusions that he has made during his many years of association with the neurosurgical unit of the Richmond Hospital of Dublin.

Congenital or "full" disks may be confusing at times even to the experienced ophthalmologist. The author prefers to defer opinion in questionable cases and to recheck the patient in one week. If no change has occurred within this time, he rechecks again in two weeks and then in four weeks; if still no change develops, he feels secure in calling the disks "normal." Hypertensive disks often present a problem, and the author states that in some cases he found it impossible to determine whether the swollen disk was hypertensive, due to a space-occupying lesion, or merely congenital.

Papilledema due to tuberculous meningitis should be treated by ventriculostomy to prevent secondary optic atrophy. Refractive errors should not be allowed to cover up other pathological changes, and usually plotting of the visual fields will clarify the problem. Chronic retrobulbar neuritis is a diagnosis that should seldom be made, and the possibility of tuberculum sella meningioma should always be considered. By careful study, optic neuritis can usually be differentiated from papilledema and the patient be spared neurosurgical procedures.

In summary, in the author's opinion the careful study of the visual acuity, the appearance of the fundus, and the perimetric findings should point toward the proper diagnosis of neuro-ophthalmological problems. Angiography or other neurosurgical procedures often are required to establish such diagnosis.—*Thomas P. Kearns, M.D.*

23. *Visual Field Changes Produced by X-ray Treatment of Pituitary Tumors.* MAX CHAMBLIN. Brit. J. Ophth. 42:193–211, April, 1958.

The author is associated with the Albert Einstein College of Medicine and the Montefiore Hospital of New York City. A study was made of 40 patients with

pituitary tumors treated with roentgen rays only. Thirty-seven patients had chromophobe adenomas and 3 had eosinophilic adenomas. Adequate visual field studies were available in all cases.

The visual fields were plotted with 2/330 targets on the perimeter and 1/2000 targets on the tangent screen. During treatment, the patients were rechecked if possible within the first 24 hours and then at intervals of three to four days. The average treatment was a total tumor dosage of 3000 r usually given in three courses of 1000 r over periods of four to six weeks with intervals of three to four weeks between.

An elaborate and, by necessity, a somewhat complicated system of statistically evaluating the changes in the central acuity and the visual fields is described. The author admits that his series of patients was small, but he expresses the opinion that the following conclusions can be drawn: (1) Patients with pituitary adenomas that are not too far advanced or do not have cystic changes show excellent response to roentgen-ray treatment as the only form of treatment. 2. A slow program of roentgen-ray treatment guided by frequent visual examinations was found to be best. 3. Improvement was noted in half, or 20, of the 40 patients. Ten patients showed improvement during treatment, while the other 10 showed improvement during the next few months or years. Four to six months should elapse after treatment before concluding that no improvement will occur, and final judgment of the total improvement should not be made for at least two to three years.—*Thomas P. Kearns, M.D.*

24. *Acute Visual Failure as a Neurosurgical Emergency.* ALFRED UIHLEIN. Brit. J. Ophth. *42*:157–172, March, 1958.

Dr. Uihlein, a neurosurgeon at the Mayo Clinic, calls attention to the fact that a lesion involving the optic pathways may be responsible for acute loss of vision. Five cases are reported to illustrate the difficulties of diagnosis that confront the physician when patients with sudden loss of vision without obvious cause are seen. One patient experienced sudden loss of vision due to hemorrhage into a pituitary tumor. Another patient was found to have had a hemorrhage into an hemangioendothelioma in the region of the optic chiasm. Other causes were astrocytoma of the optic nerve, aneurysm of the internal carotid artery, and chiasmal neuritis.

To prevent overlooking such lesions and to prevent irreparable loss of vision, the patient should have a thorough medical examination, which should include ophthalmological examination, roentgenograms of the skull, neurological examination, and accurate plotting of the visual fields. In more difficult problems, further neurosurgical procedures such as pneumoencephalography and cerebral angiography may be required.—*Thomas P. Kearns, M.D.*

25. *Horner's Syndrome: An Analysis of 216 Cases.* C. L. GILES AND J. W. HENDERSON. Am. J. Ophth. *46*:289–296, Sept., 1958.

The most common site of interruption of the sympathetic pathway to the eye is in the sympathetic trunk and in the postganglionic fibers before they course intracranially. The spinal roots are affected less than half as often as the sympathetic

trunk, while other sites are only rarely affected. Neoplasia, malignant far more often than benign, is the most common etiological agent. Bronchogenic and metastatic carcinoma are the most frequent malignancies, and neurofibroma and thyroid adenoma are the only significant benign tumors. Less frequent causes are surgical procedures, nonoperative trauma, and vascular disease. Herpes zoster was the only lesion implicated in an intracranial postganglionic lesion. In view of the frequently serious nature of the lesions involved in the production of a Horner's syndrome, a complete investigation is warranted.—*Ralph Z. Levene, M.D.*

MEDICAL OPHTHALMOLOGY

26. *Ocular Lesions of Temporal Arteritis.* H. P. WAGENER AND H. H. HOLLENHORST. Tr. Am. Ophth. Soc. 55:249, 1957.

One hundred and twenty-one patients with temporal arteritis were studied at the Mayo Clinic, of whom 54 had some degree of visual loss. The most common cause of loss of sight was active ischemia of the optic nerve, affecting one or both eyes in 33 of the series. Permanent loss of vision is sometimes preceded by transient blurring. Other changes observed were ischemic change in the retina, infection of the retina, ischemia of the extraocular muscles. Palpation of the temporal arteries and sedimental rate of red blood cells aid in diagnosis. Biopsy of a segment of the temporal artery is valuable. Adrenal corticosteroids will reduce the incidence of blindness.—*Eugene M. Blake, M.D.*

27. *Early and Delayed Ocular Manifestations of Congenital Toxoplasmosis.* M. J. HOGAN, S. J. KIMURA, ANN LEWIS, AND PHYLLIS A. ZWEIGART. Tr. Am. Ophth. Soc. 55: 275, 1957.

This disease was first recognized in 1938 and it was apparent that the most important manifestations of the disease were in the central nervous system and the eye. The methylene blue test is a great aid in diagnosis. The disease occurs in acute and subacute forms depending on the stage of pregnancy when infections occur. Lesions are bilateral in 80 per cent of cases. The skin test cannot be depended on for diagnosis of the congenital form. Skull roentgenograms are of little value. Pyrimethamine, sulfathiazole, and prednisolone were employed in treatment.—*Eugene M. Blake, M.D.*

28. *Leprosy of the Eye.* WILLIAM JOHN HOLMES. Tr. Am. Ophth. Soc. 55:145, 1957.

This very complete and instructive picture of the effect of leprosy on the eye, by an acknowledged authority, should be read in its entirety. The disease affects more than five million persons and is most frequent in tropical and subtropical countries; males are more predisposed to the disease.

The eyes are especially prone to involvement, particularly where there is general debility. In the lepromatous form of infection, the ocular lesions are due to destruction of healthy tissue and replacement by a granulomatous type. Every portion of the eye may be involved, including bony changes in the orbit and lachrymal apparatus, the eyebrows and eyelids. The conjunctiva is rarely involved, but the

organisms are often found in the secretion. The episclera and cornea are very vulnerable and the iris and ciliary body frequently infected, while destruction of the posterior segment is rarely seen.

The therapy of leprosy is chiefly the sulfone drugs, and especially if employed before destructive change develops. Steroids and chaulmoogra oil are not so effective. To quote the author, "with adequate sulfone therapy and timely and appropriate prophylaxis we can prevent much suffering and eliminate needless blindness. With proper medical and surgical management we can conserve sight and restore . . . vision to patients . . . previously considered beyond help."—*Eugene M. Blake, M.D.*

29. *Angiomatosis Retinae Eleven Years after Diathermy Coagulation.* DERRICK VAIL. Tr. Am. Ophth. Soc. 55:217, 1957.

Angiomatosis retinae (von Hippel's disease) and Lindau's disease are more common than is generally supposed. The development of the former disease is divided into four stages and the earlier changes offer the better prognosis from treatment. The author carefully differentiates the diagnostic features. Treatment has included radium, roentgen rays, catholysis, diathermy coagulation, and heliocautery. Operation is successful in 70 per cent of cases. The greatest risk in treatment is intraocular hemorrhage, and multiple tumors are better treated separately and at intervals. The literature is well reviewed.—*Eugene M. Blake, M.D.*

30. *Acute Tuberculous Endophthalmitis.* G. DVORAK-THEOBOLD. Tr. Am. Ophth. Soc. 55:325, 1957.

The patient described had been treated for six months for asthmatic attacks by injections of ragweed antigen. At the end of this period the patient developed an endophthalmitis, which progressed to destruction and enucleation of the eye. Sections showed giant cells and other changes typical of tuberculosis. Addison's disease of tuberculous form followed ocular involvement.—*Eugene M. Blake, M.D.*

31. *Observations on Uveitis Associated with Viral Disease.* P. THYGESON, M. J. HOGAN, AND S. J. KIMURA. Tr. Am. Ophth. Soc. 55:333, 1957.

This very extensive study of the relationship of uveitis to other diseases, especially viral, is based on research on 1000 patients with uveitis. Viral disease complicates herpes simplex, herpes zoster, varicella, influenza, lymphogranuloma venereum, and infectious mononucleosis. Herpetic uveitis may occur without keratitis, and is probably of virus origin, and with Vogt-Koyanagi disease, Harada disease, and sympathetic uveitis. The significance of serological tests, skin tests, and cytological studies of aqueous and uveal tissue is reported.

This extensive study should be read in its entirety.—*Eugene M. Blake, M.D.*

32. *Optic Neuropathy of Grave's Disease.* J. W. HENDERSON. Tr. Am. Ophth. Soc. 55:353, 1957.

Six cases personally observed and 10 others reported in the literature of a seldom

recognized disease of the optic nerve are described. The disorder is manifested by the demonstration of either central scotomas or defects of the nerve-fiber bundle on examination of the visual fields. The clinical course of the disease is much like that of retrobulbar neuritis. To differentiate the two conditions, the name of optic neuropathy of Grave's disease is proposed. The course of the disease is not affected by treatment of the thyroid or its association with the pituitary, but the prognosis is good in most cases.—*Eugene M. Blake, M.D.*

33. *Toxoplasmosis: The Nature of Virulence.* H. E. KAUFMAN, J. S. REMINGTON, AND L. J. JACOBS. Am. J. Ophth. *46*:255-261, Nov., pt. 2, 1958.

Utilizing the invasiveness and multiplication rate of *Toxoplasma* organisms in monkey kidney tissue culture cells as a measure of virulence, the authors investigated three different strains. They demonstrated a positive correlation with the usual virulence criterion of per cent mortality in animals given graded inocula of this parasite. The determination of differences in multiplication rates may be important in influencing the efficacy of present anti-*Toxoplasma* chemotherapy. In general, the slow-growing strains are believed to be more resistant to metabolic antagonist drugs. Preliminary investigation on the effects of pyrimethamine support this hypothesis. —*Ralph Z. Levene, M.D.*

THE CORNEA IN OPHTHALMOLOGY

34. *Familial Corneal Dystrophy Lattice Type.* R. M. RAMSAY. Tr. Am. Ophth. Soc. 55:701, 1957.

Before the advent of the biomicroscope there was great confusion in the nomenclature of congenital corneal changes of the familial type. The etiology is unknown, but several theories have been advanced. A case is here reported in detail. The disease is usually bilateral; transmission is dominant and not sex-linked. Consanguinity is often recorded and the etiology is undetermined.—*Eugene M. Blake, M.D.*

35. *Clinical Observations on the Treatment of Bullous Keratopathy.* L. V. JOHNSON. Tr. Am. Ophth. Soc. 55:543, 1957.

The author discusses the clinical deviation from the normal corneal endothelium and the pathological physiology of these deviations. Clinical observations and the treatment are discussed. Bullous keratopathy appears to be the result of altered salt and water transfer. Neurectomy of the greater petrosal nerve and action of roentgen rays frequently result in a relatively dry surface of the eye.—*Eugene M. Blake, M.D.*

36. *Artificial Corneal Implants.* ROBERT DAY. Tr. Am. Ophth. Soc. 55:455, 1957.

An interesting review of the literature shows clearly that the exposed penetrating transplants fail to afford permanent improvement in vision. Failure results chiefly from eventual separation of the prostheses due to infection, epithelial downgrowth,

or the formation of an opaque membrane on the back surface of the prosthesis.—*Eugene M. Blake, M.D.*

37. *Incorporation of S^{35} in Healing Wounds in Normal and Devitalized Corneas.* J. H. DUNNINGTON AND GEORGE K. SMELSER. Tr. Am. Ophth. Soc. 55:67, 1957.

The normal cell constituents of corneal tissue adjacent to an injury are most important in the process of healing. If cells are destroyed by freezing, the ground substance is not renewed at a normal rate and it is possible that inflammatory cells break down the ground substance. The latter is the new connective tissue element found in wound healing. A delay of three or four days in healing occurs if the cornea is temporarily devitalized.—*Eugene M. Blake, M.D.*

38. *Corneal Changes from Hydroquinone Exposure.* BANKS ANDERSON AND FRANK OGLESBY. Tr. Am. Ophth. Soc. 55:33, 1957.

Workers who are long exposed to the fumes of hydroquinone are prone to develop staining of the cornea and conjunctiva. The slit lamp shows the discoloration to be due to the deposition of dark brown granules within the conjunctiva or in Bowman's membrane. A striking change is the development of Hudson-Stähli lines with a flattening of the cornea and a resultant high degree of astigmatism.—*Eugene M. Blake, M.D.*

39. *The Potential Value of Human Tear Patterns.* P. ROBB MCDONALD, IRVING H. LEOPOLD, AND THEODORE W. SERY. Tr. Am. Ophth. Soc. 55:49, 1957.

In this study, filter paper strips employing the Spinco R model were used. The components of the tears are a distinctive protein, lysozyme, a gamma globulin, and intermediary globulins. Tear patterns in normal eyes are similar and the same in each eye. The patterns tend to be reproducible in both eyes. The patterns vary in proportionate distribution at different times of the day in normal eyes but the two eyes are similar. Keratoconjunctivitis sicca leads to a reduction of lysozyme content. Uveitis may show alteration of tear pattern in the affected eye.—*Eugene M. Blake, M.D.*

40. *Penetrating Keratoplasty.* B. D. LEAHEY. Tr. Am. Ophth. Soc. 55:575, 1957.

In this comprehensive paper the author gives the complete outline of the subject of corneal grafting. Both lamellar and penetrating grafts were employed in a series of 148 eyes operated on. Stress is laid on preparatory procedures in this type of surgery, such as elimination of blood vessels, improvement in fixation of the graft, prevention of posterior adhesions, and supportive medication, especially steroids. The article is illustrated with 25 figures.—*Eugene M. Blake, M.D.*

41. *The Role of Beta-ray Irradiation in Antibody Formation in the Rabbit's Cornea.* J. I. MOORE. Tr. Am. Ophth. Soc. 55:637, 1957.

The purpose of the investigation was to study the effect of beta-ray irradiation on

the formation of antibodies in the rabbit's cornea. The irradiation was known to lessen the inflammations of the anterior segment. While the local formation of antibodies in the corneal tissue is not proved by this work, the change in the appearance of the corneal tissue, in which the reaction was brought about by the injection of antigen, makes an interesting subject for speculation and further investigation.—*Eugene M. Blake, M.D.*

42. *Lipid Keratopathy and Atheroma.* DAVID G. COGAN AND TOICHIRO KUWABARA. Circulation *18*:519–525, Oct., pt. 1, 1958.

A marked anatomical and histological similarity is demonstrated between fatty plaques of the cornea of man and of hypercholesteremic rabbits. These plaques appeared in an area of the cornea that had previously been vascularized. The process of atheroma formation in the intima of blood vessels of man and hypercholesteremic rabbits also shows many features of fatty plaque formation in the cornea. This process consists of intracellular globular sudanophilic lipid, which is associated with cellular hyperplasia, and extracellular granular sudanophilic lipid, associated with hyalinization and necrosis of tissue. This latter process is associated with the release of birefringent crystals suggestive of cholesterol. Prior vascularization must be present in all cases.

Since lipid plaques of the cornea occur in a manner similar to atheroma of blood vessels, atheroma formation can be studied in terms of the analogous situation of the cornea, which had the advantage of being visualized directly.—*Carl Kupfer, M.D.*

BOOK REVIEWS

So You Have Glaucoma. EVERETT R. VEIRS. New York, Grune & Stratton, Inc., 1958. Pp. 64. Price $2.75.

According to the author, the purpose of this book is to present to the patient who has glaucoma information that will give him a better understanding of his condition. This is a valuable contribution in the important matter of education of the public. The author is to be congratulated on writing such an informative book. How well Dr. Veirs has succeeded in writing an interesting, factual book can be realized only by reading his instructive chapters on the history of glaucoma, its frequency, anatomy, and physiology, types of cases, signs and symptoms, and diagnosis and treatment.

In the final chapter, called "Postscripts," he has the following summary of "Do's": Use medication faithfully, return for periodic check-ups, see your doctor about pain, tell the family physician you have glaucoma and in changing physicians, transfer your records, and finally, follow the ophthalmologist's instructions.

This book is exceedingly valuable not only to the patient who has glaucoma, but also for the general physician who wishes to know more concerning glaucoma without reading a voluminous text. The material is clearly and interestingly presented without too much detail, but with great scientific accuracy. The author points out the need for having the general physician know that his patient has glaucoma so

that he may avoid the use of atropine and its derivatives unless he has the consent of the ophthalmologist.

No mention of taking tension for screening purposes by general physicians, with the ordinary tonometer or the hypertension indicator, is made by the author. This is probably because the book was written primarily for the patient. However, as patients are beginning to question general physicians about taking the tension, it seems unfortunate, although understandable, that this was not mentioned.

I recommend this book with enthusiasm to patients who have glaucoma, to laymen and nurses who wish to know more concerning glaucoma, and to general physicians, who should do more in finding the 2 per cent of undetected glaucoma in their patients older than 40 years.—*Conrad Berens, M.D.*

History of Ophthalmology Published

A History of Ophthalmology by George E. Arrington, Jr., M.D., was recently published by MD Publications, Inc., as part of their series of *MD Monographs on Medical History*. The ophthalmology monograph is a study of the development of the "ophthalmological mind" throughout the history of civilization. While the achievements of therapeutic and diagnostic techniques are cited, it is their relationship to concepts of natural science, biology, evolution, philosophy, and human culture generally that is emphasized.

The book attempts to guide the ophthalmologist to the sources of concepts that regulate his daily clinical functioning. He is introduced to the great thinkers who established through both reasoning and experimentation the basic ideas of visual function and dysfunction on which the science of ophthalmology is founded. Ophthalmology is carefully related to medicine generally, and their roles of interdependence and interaction are shown.

Dr. Arrington is Associate in Ophthalmology, Medical College of Virginia, Richmond, and Attending Ophthalmologist, Medical College of Virginia Hospital, Richmond Eye Hospital, Retreat for the Sick Hospital, and the Richmond Memorial Hospital.

Isomer of Vitamin A May Be Answer to Retinal Degeneration

VITAMIN A--ESSENTIAL IN NIGHT VISION. It has long been known that vitamin A is an essential factor in the maintenance of the retinal pigment and in night vision. The ability to see well under reduced illumination (such as moonlight) is dependent on the formation in the retina of rhodopsin from vitamin A.

COMMON FORM OF VITAMIN A OF LITTLE BENEFIT. Wald demonstrated that the commonly administered form of vitamin A (so-called all-trans form) will not be directly converted to rhodopsin by the human body. Oroshnik carried this one step further and demonstrated that the necessary ingredient is the 11-cis form of vitamin A. This fact was borne out in laboratory animals. Newborn cats fed on all-trans vitamin A underwent retinal degeneration. When similar animals were given the 11-cis form, there was no retinal change.

METABOLIC FAILURE RESPONSIBLE FOR RETINAL DEGENERATION. It is now believed that the human body must isomerize the 11-cis vitamin A from the all-trans form in order to maintain the integrity of the retinal pigment. Failure in this metabolic process may be the underlying factor in familial retinal degeneration, known as retinitis pigmentosa.

G. Peter Halberg, M.D.
New York, N. Y.

Sixth Pan American Congress of Ophthalmology to Meet in Venezuela in 1960

The Sixth Pan American Congress of Ophthalmology will be held in Caracas, Venezuela, Jan. 31-Feb. 7, 1960. The scientific program will consist of round table discussions, symposia, free papers, motion pictures, scientific and technical exhibits.

Those who are to participate in the program are urged to send titles and short abstracts of papers and applications for the showing of new films and exhibits before May 1, 1959, to Dr. James H. Allen, New Orleans, chairman of the program committee for North America. Ophthalmologists north of Panama wishing to participate may write to Dr. Allen at Tulane University School of Medicine, 1430 Tulane Avenue, New Orleans 12, Louisiana.

CASE REPORT

Closed-Angle Glaucoma

A 66-year-old housewife was seen in the office complaining of episodes of pain and blurred vision in the eyes of three months' duration. She stated that she occasionally awakened in the morning with pain in the eyes, especially the left eye. Her past medical history, family history, and systems review were noncontributory. The visual acuity in the right eye was correctible to 20/20 and in the left eye to 20/30. With the proper reading addition, she could read the finest newsprint. She had some opacification of the left lens. The left optic nerve was somewhat pale and appeared slightly cupped. The ocular tension in each eye with the pupil undilated and dilated was 28 mm. of mercury Schiøtz (normal, 15 to 25 mm.). Visual fields were performed and revealed characteristic glaucomatous defect. Visualization of the angle of the anterior chamber revealed adhesions of the iris to the chamber angle, blocking Schlemm's canal.

A diagnosis of chronic closed-angle glaucoma was made. The patient was placed on 2 per cent pilocarpine, four times a day in each eye. An operative procedure was advised, but the patient was reluctant to undergo surgery.

The patient was seen again six days later at which time she complained of severe pain in the left eye, poor vision, nausea, and vomiting. Examination revealed a bullous breakdown of the corneal epithelium. The ocular tension was more than 90 mm. of mercury Schiøtz. A diagnosis of acute narrow-angle glaucoma was made and the patient was hospitalized. The tension in the left eye was 16 mm. Schiøtz within three hours after the use of 5 per cent neostigmine ophthalmic drops every 30 minutes and 500 mg. of acetazolamide intramuscularly. After this a filtering operation (iridenclysis) was performed. Two days later a prophylactic, peripheral iridectomy was performed on the right eye.

The patient had an uneventful convalescence. Visual acuity in the right eye and the left eye was unchanged from the initial examination. Tension was normalized in each eye. Unless there is a medical contraindication, chronic closed-angle glaucoma requires immediate glaucoma surgery. Delay in surgery leads to a poorer prognosis because of continuation of angle closure and the real possibility of an acute attack of glaucoma. The selection of the type of glaucoma surgery to be performed is dependent on the amount of angle closure.

Dabar Cury, M.D.
Huntington Park, Calif.

EDITOR'S NOTE: This section is devoted to case histories of typical daily problems in the eye field. It is designed to help the reader toward a better understanding of diagnoses and treatment in ophthalmology.

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MD MONOGRAPHS ON MEDICAL HISTORY NO. 2

A History of Ophthalmology

by GEORGE E. ARRINGTON, JR., M.D.
Associate in Ophthalmology
Medical College of Virginia, Richmond, Virginia

Foreword by FELIX MARTI-IBAÑEZ, M.D.

A History of Ophthalmology presents a study of the development of the "ophthalmological mind" throughout the history of civilization. The book attempts to guide the ophthalmologist to the sources of concepts that regulate his daily clinical functioning. He is introduced to the great thinkers who established, through both reasoning and experimentation, the basic ideas of visual function and dysfunction on which the science of ophthalmology is founded. Ophthalmology is carefully related to medicine generally in this work, and their roles of interdependence and interaction are shown. 192 pp., clothbound, price $4.00.

A History of Ophthalmology is the second book in a series written and designed for physicians, sociologists, educators, and members of related professions, as well as residents, interns, and students. Under the editorial direction of Félix Martí-Ibáñez, M.D., Professor and Director of the Department of the History of Medicine, New York Medical College, Flower and Fifth Avenue Hospitals, the series is designed to enrich the physician's knowledge of medicine and to impart useful information. *A History of Public Health* by George Rosen, M.D., Ph.D., M.P.H., was recently published. Soon to appear is *A History of Neurology* by Walther Riese, M.D. Beautifully designed and attractively bound in durable cloth, *A History of Ophthalmology* exemplifies the ultimate elegance in the art of bookmaking and will make a handsome addition to your library.

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